

MEDICAL ASPECTS OF OCCUPATIONAL DISEASES

THOMAS F. MANCUSO*

- I. Introduction
 - A. Scope of Occupational Diseases
 - B. Occupational Exposures
- II. What is an Occupational Disease?
 - A. Definition
 - B. Association
 - 1. Acute Illness
 - 2. Chronic Illness
 - 3. Difficulties of "Association"
 - C. Symptoms of Occupational Diseases and Common Diseases
 - D. Variations in Exposures and Population Groups
 - E. Diagnosis of Occupational Diseases
- III. Statutes of Limitations
 - A. Occupational Diseases
 - B. Silicosis
 - C. Berylliosis
- IV. Lung Diseases Due to Dust
 - A. Silicosis
 - B. Asbestosis
 - C. Alumina Abrasives
 - D. Other Lung Diseases
 - E. Pulmonary Emphysema
 - 1. Disability
- V. Radiation Hazards
- VI. Reference Data on Specific Substances
 - A. Toxic Substances, Occupations and Symptoms
 - B. Threshold Limit Values, American Conference of Governmental Industrial Hygienists

INTRODUCTION

In participating in this symposium, I have two objectives: (1) to meet the requirements to provide essential data on the medical aspects of occupational diseases within the purview of the laws of workmen's compensation, and (2) to utilize this opportunity to broaden the knowledge of attorneys concerning the scope of occupational illnesses and

*Mancuso, Thomas F., M.D., M.P.H., Ohio Department of Health, Chief, Division of Industrial Hygiene.

provide them with some understanding of the chemicals and occupations involved in the industries of Ohio. These principal objectives will be interwoven as a combined approach in the data. The facts that follow will be presented purely from the medical viewpoint.

Since this symposium represents a significant means to reach all the attorneys within the state and provide them with information about occupational diseases, a particular pattern of presentation has been selected to facilitate this purpose and provide ease in reference. Naturally, there are limitations as to how much material can be included at this time. However, I shall try to strike a balance between information desired by the attorneys who are casually acquainted and attorneys who are familiar with this field; also, to provide sufficient new data to make it interesting even to the attorneys specializing in workmen's compensation. Portions of the material utilized have already been presented to all the physicians in Ohio in a similar effort in order to provide a greater understanding of occupational diseases.

Scope of Occupational Diseases

In Ohio the industrial population comprises some 2,300,000 workers who are potentially subjected to occupational diseases and industrial public health hazards resulting from exposure to toxic substances in their working environment. These diseases may be of a serious nature affecting the various vital organs and systems of the body, depending upon the nature of the toxic dusts, fumes, mists, vapors, gases or other substances and the concentrations involved. In general a broad classification of the occupational diseases due to physical and chemical agents may be grouped into the following four categories: (1) toxic dusts, mists, fumes, vapors and gases; (2) physical states of the environment such as abnormal temperatures and pressures, radiant energy and noise; (3) mechanical factors such as vibration, pressure, physical strain and movement; and (4) infectious agents connected with industrial occupations.

Chemicals may be absorbed through the skin, inhaled through the respiratory tract or ingested due to contamination at work from improper work processes or due to lack of adequate cleaning facilities and personal protective measures. Chemicals in general vary in their toxicity; their effects may be acute or chronic and may affect one or more organs at different times. We know, for example, that silica, asbestos and beryllium produce changes in the lungs with physical disability of long or permanent duration. Benzol, toluene and other solvents produce diseases of the blood and blood-forming organs. Carbon tetrachloride, tetrachlorethane and other halogenated hydrocarbons produce toxic effects on the liver and kidneys. Manganese, mercury and carbon disulphide affect the brain and central nervous system.

Some chemicals may produce specific cancers such as cancer of the

bladder due to aromatic amines. Destructive changes in the bone have occurred due to arsenic and phosphorus. Perforations of the nasal septum as well as lesions of the oral cavity result from excessive exposure to chromium, alkalis and acids. The gastrointestinal tract is affected symptomatically by chemicals capable of acting as systemic poisons.

Gases such as carbon monoxide, phosgene and cyanogens cause asphyxiation, while other gases such as nitrous oxides produce chemical pneumonias. Exposure to x-rays and emanations from radium may produce serious burns and cancer while the blood and blood-forming organs are also profoundly affected by radiation. Infrared rays may produce cataracts; and methyl chloride, blindness.

Chemicals which act as primary skin irritants or as skin sensitizers cause occupational dermatoses, the most frequent of the industrial diseases.

Occupational Exposures

The magnitude and complex nature of the occupational disease problem is reflected in the number, variety and location of industries in the state. There are over 12,500 manufacturing industries utilizing hundreds of varied chemicals and materials which require exposure of individuals to toxic dusts, fumes, vapors, mists, gases, etc. which are evolved during the manufacturing processes. Ohio leads the nation in the production of steel and glass and in the number of foundries as well as electrical appliances and the ceramic arts. The state is noted for its wide variety of manufactured products and industries in all sections of Ohio.

Our state has a rather remarkable distribution of industries, both large and small, within its various counties and there is hardly an area which does not have some form of industrial employment. In this respect practically every physician and attorney has an opportunity within the area he lives and practices to observe individuals who have been afflicted with occupational illnesses. Even agriculture presents important occupational health problems through the wide-spread use of chemicals in the spraying of crops and the various insecticides. Lead, arsenic, nicotine and DDT have been commonly recognized but the recent increasing use of organic phosphates such as "parathion"¹ and "TEPP" (tetraethylpyrophosphate) have been dramatic and deadly in their toxic effects. Occupational diseases then may occur under a variety of conditions and in a tremendous number of different environmental exposures.

Each type of industry and individual plant poses specific industrial health problems. In each instance of occupational disease, there are medical and engineering control measures which can prevent the occurrence of additional cases of illness and can control the extent of the toxic exposures.

¹ SAX, DANGEROUS PROPERTIES OF INDUSTRIAL MATERIALS (1957).

WHAT IS AN OCCUPATIONAL DISEASE?

Definition

The Workmen's Compensation Act gives the following definition of an occupational disease: "a disease peculiar to a particular industrial process, trade, or occupation and to which an employee is not ordinarily subjected or exposed outside of or away from his employment."²

This brings us to the question, what are the problems associated with the interpretation of this definition? Evidently they are related to the clarification of the word "peculiar" as it differs from the usual or normal. In determining whether a disease is "peculiar" to a particular process or an occupation, there must be an "association" of factors, and, in turn, an understanding of the difficulties involved. Occupational diseases do not "normally" exist, but rather they represent an incidence of disease that occurs because of the very nature of the employment or environmental exposure. Silicosis, for example, identified as an occupational disease, must be shown to be "peculiar" to the occupation—does not occur in the general population—but is found only in certain occupational exposures. Workers with silicosis have the same "unusual" chest film, have symptoms peculiar to silicosis cases and previous exposure to silicon dioxide. In comparison, in order to see whether this is "peculiar" or important in these workers, the observations are sought in the general or control populations. In this manner the disease is determined to be "peculiar" rather than a "normal" occurring phenomenon.

Association

In the interpretation of occupational diseases the word "association" is basic. If certain factors in the working environment are consistently associated with the development of a specific disease or syndrome, then the basis is established for the consideration of an occupational disease. Association of factors from observations is manifest daily in the practice of medicine especially when one correlates symptoms and physical findings in the examination of a patient by a physician. The physician has learned to "associate" the disease entity with the symptoms as proven by earlier experience or pathological reports. Similarly, the attorney learns to "associate" certain factors and circumstances as evidence in the establishment of a particular case. The strength of the evidence bears a direct relation to the strength of the "association," the links from one event to another.

In order to determine what constitutes a clue or a valid "factor" associated with an event, it is necessary to know the "normal" or range of variation of the factors whether at the scene of a crime or at an industrial scene. Similarly, we must know the range of variations of the disease according to the characteristics of the populations involved and

² OHIO REV. CODE § 4123.68(X) (1953).

the factors associated with the disease. Perhaps a few illustrations will help clarify this point.

Acute Illness

We have as an example an event in a small town where invitations were extended to attend a community supper. The meal was served on a Saturday evening and four to eight hours later the people who attended became ill with nausea, diarrhea and vomiting. The doctor who was called to many of the homes recognized the symptoms of food poisoning. He discovered that all of the individuals had the same symptoms, all ate the same food at the same place and at the same time. The doctor *associated* the cause of the disease with the eating of this particular meal.

Now let us look at an example of "association" relating to industry. We find workers cleaning out a tank with a solvent called carbon tetrachloride in a room that is not properly ventilated. A number of workers engaged in the same process at the same place become ill with the same symptoms and collapse. Since their work process and the use of carbon tetrachloride were the common factors in their clinical history, it is evident their illness is directly *associated* with their work.

The two illustrations of "association" which have been described are immediately observed because they relate to an *acute* incident producing an acute disease, *i.e.*, the development of a disease or illness from a few hours to one or two days following exposure.

Chronic Illness

Let us now consider what is known as the chronic type of exposure.

Some 200 men worked in a plant which manufactures chromates, and about twelve to fifteen years later a significant group of these men developed lung cancer and died. In another plant where dyes were manufactured, observations showed that deaths some twenty or more years later from bladder-cancer among the employees were far greater than one might expect. In neither case did the workers, their families or their physicians "associate" this particular disease or death with employment because of the so-called latent period, the long time it takes between occupational exposure and the actual development of the disease. This type of exposure then is frequently described as "chronic" in which the development of disease and symptoms of disease may be delayed weeks, months or years after the occupational exposure occurred.

Difficulties of "Association"

There are difficulties too in this matter of "association." We realize there are hundreds of chemicals being used and introduced in various combinations in industry which may cause subsequent serious effects on the human body and that symptoms of disease may often develop months

or years after the critical exposure. In addition, the last place of employment is not necessarily the work place where the initial exposure occurred. Individuals may move from job to job and the recognition of an "association" of such an exposure and the detection of the chronic disease which may develop requires comprehensive work histories and some knowledge of the substances used in various industries.

Symptoms of Occupational Diseases and Common Diseases

Certain symptoms are common to many diseases, industrial diseases or the general common diseases, although the causative factors may be different. For example: the abdominal pain of acute appendicitis and that of lead poisoning may be very similar; the changes in the central nervous system due to manganese poisoning produce symptoms that are remarkably like those of multiple sclerosis; similarly, the psychosis produced by carbon disulphide exposure is difficult to differentiate from the psychosis due to some other cause; aplastic anemia may be due to benzol, TNT or some other unidentified cause; the same is true for leukemia. Individual symptoms, therefore, in general, are not identifiable as being solely peculiar to a given substance. A most common example would be that of headache which could have a variety of causes, both occupational and nonoccupational. For some diseases, however, such as silicosis, a chest x-ray may provide a certain characteristic picture. The same may be said of berylliosis although the differential diagnosis is much more complex. However, the number of occupational diseases which can be identified and detected by such objective means are markedly limited.

Considering the entire field of occupational diseases, one must realize that the symptom complexes may bear striking appearances to non-occupational syndromes and that interpretation of whether an occupational disease exists or not should not be based solely upon the symptoms involved. It is true, however, that there are some clinical occupational syndromes, such as those associated with mercury, which can make one seriously suspect an occupational disease. The point I want to emphasize, however, is that although symptoms *may* be "peculiar" they do not necessarily *have* to be "peculiar," to still fall into the category of a true occupational disease. The recognition of these symptom complexes evolves when the consideration of the working environment reveals the symptoms are associated with a particular procedure, operation or working exposure, just as the physician has learned from his pathologist reports, that the clinical symptoms initially observed are associated with a specific disease entity. Such symptom complexes have occurred and observations have been made on both humans and animals subjected to the toxic effects of certain substances.

The particular pattern of symptoms which has been observed following exposure to specific substances such as lead has been derived

from the identification of that particular occupational disease as confirmed by medical and engineering studies and by toxicological experiments. This is true of a significant number of toxic substances. However, there are marked gaps in our present knowledge of the literally hundreds of chemicals being used in industry in various combinations for which no toxicological data is available. As a consequence it makes diagnosis extremely difficult for a physician to recognize the occupational diseases due to these substances as well as the new chemicals introduced in industry because of the newness of the exposure and the lack of previous human exposure experiences in industry. This was true when toluene di-isocyanate (TDI) was introduced in industry and there were very few actual human exposures. Following its introduction in Ohio an outbreak occurred in various different plants in which this substance was used. A few of the employees developed asthmatic-like symptoms with cough and pain in the chest. Since the operation was a small one, only a few employees were affected in each plant. The symptoms of asthma that developed were not much different from the normal symptoms of asthma but the disease became "peculiar" because of its direct association to a specific exposure to a process, TDI, which became all the more remarkable since the amount necessary to produce a toxic effect on the worker was only one or two ppm (parts of the substance per million parts of air).

Equally important information about this particular substance, TDI, is the fact that it is a strong sensitizing agent and a worker acquires an allergic response to the chemical. Thus, when a worker enters the area in which the substance is used, he develops symptoms and is not able to perform his work. The sensitivity which the worker develops may be of such a high degree that an individual can no longer be exposed in operations in which this chemical is used because of the asthmatic-like type of response which recurs.

One of the differentiating points of an occupational disease, in those other than the dust diseases, is the fact that an individual worker usually improves when he is removed from his exposure and the condition recurs or is aggravated on re-exposure. In cases of poisoning, the disease is most frequently chronic rather than acute.

Variations in Exposures and Population Groups

A disease may be peculiar to a particular industrial process and be initially detected by an abnormal incidence, *i.e.*, a greater number of cases of the same illness within a group of workers exposed to the same operation. The most frequent example of this would be dermatitis where a group of employees are exposed to skin irritants or sensitizers and a large percentage of workers develop dermatitis on the same area of the body. However, the question of "peculiar to environment" should not be based solely on the relative percentage of workers affected, although this is an important factor, because there are marked differences in the working

environment, the lengths of employment and the susceptibility of various workers. The composition of the work force in terms of age, sex, and color represent factors to be considered, depending upon the environmental exposures involved. Also, the nature of the operations may require that only one person within a group carry out certain work procedures and be exposed to a particular chemical. If this worker develops a systemic or respiratory disease actually due to the inhalation of a chemical, the fact that he was the only person to develop the disease should not detract from the validity of his claim. It may be more difficult to interpret and prove the association; nevertheless, the occupational disease can occur and does exist.

In the study of population groups in attempting to determine whether a disease is appearing at a rate greater than "usual" or "normal," it is important to take into consideration the length of exposure, that is, the employment exposure in a particular department or process. A plant may have a procedure of rotating employees exposed to a toxic substance, benzol for example, where the procedure frequently has been to perform blood counts every month on these employees, and when the blood count shows some change suggestive of a toxic effect to move the employee affected to another department. In this practice a larger number of employees may receive exposure whereas a smaller number would be involved in any prolonged exposure. A survey of the department may not convey a true picture of the actual number of employees who had been affected. Similarly, if so many exposure years are required for a disease to develop, the survey of employees should take this point into consideration.

The labor turnover in a particular plant or particular department as well as sick leave policy may also be such that a true picture of what occupational diseases have occurred in the plant becomes difficult. In some instances persons with an occupational disease have been pensioned or placed on sick leave and no occupational disease claims filed.

An employee may sustain an occupational disease by the very location of his particular work procedure rather than to the actual operation itself, that is, adjacent to some other toxic exposure. In such instances the illness may occur in a department which does not have the same toxic substance but the ventilation is such that the fumes pass into the breathing zone of the worker located in the adjacent department. This then becomes peculiar to the worker exposed in the sense that his work requires him to be at that particular location even though his particular process does not generate the toxic substance. Similarly, there must be proper recognition of the disease itself, that is, the syndrome or pattern that may be recognized as being similar or identical to a disease associated with a specific substance. One must look for the factor, the toxic substance with which the disease has been previously associated and identified, for example, beryllium and silica. However, should there be difficulty in locating the factor, this should not deny the existence of the already identified occupational disease. The

many changes in plant processes and working procedures, changes in production schedules, substitution of chemicals, etc. prevent or complicate the tracing of the likely causative factor which becomes an intricate detective-like procedure.

An individual is not ordinarily subjected or exposed to most chemicals outside or away from his employment; however, the trend to greater home use for some of these in the form of repair and cleaning purposes, as well as in the use of hobbies, create a situation in which similar chemicals may be used in both the working and home environments. This introduces another problem, that of quantity or concentration of the chemicals in terms of parts per million (ppm), milligram per cubic meter (mgm./cu.mtr.), etc. of the toxic substances and the duration of the exposure (see Threshold Limits Section-ACGIH).

The workers' amount of exposure is tremendously greater in industry in terms of quantity and duration. The concentration of a given substance may vary by process and by department within the same industry. Even a breakdown in ventilation or some similar action may produce an entirely different situation in a matter of minutes in terms of concentration.

At this point I should like to mention that in the investigation of a case of silicosis or any dust disease, the dust counts at the time of the investigation, the percentage of silica or chemical involved, particle size of the dust, etc. may have no bearing whatsoever as to the true exposure of some five or ten years previously. In other words, today's investigation report of environmental conditions cannot possibly be the same as prevailed some five to ten years ago. Installation of control measures, ventilation systems, etc. may reveal that the dust counts are very low and the parting compound in the foundry contains no free silica whereas actually the opposite was the case. In fact, the parting compound could very well have contained 100 per cent free silica in previous years. The investigations, therefore, in determining the toxic factors associated with the disease syndrome must relate it to the years of actual exposure and not just to the current or prevailing situation. Admittedly this is difficult but the complex nature of an occupational disease warrants such a thorough and careful appraisal.

Diagnosis of Occupational Disease

In establishing whether a disease is occupational or not the physician makes that decision. This requires that the physicians involved have an extensive knowledge of the environmental factors which produce disease and illness and are aware of the toxic substances which are used in different industries and occupations. Unfortunately, relatively few physicians have a personal acquaintance with the working environment of their patients and knowledge of the toxicological effects of the wide-range of chemicals used in various occupations. The employee or patient is even less likely to be acquainted with the harmful nature of the substances to

which he is exposed even though he is required to specify this in his application claim for occupational disease compensation.

Basically, in order to determine whether there is any relation between the symptoms and occupation, it is necessary for the physicians to obtain an accurate occupational history from the workers who become their patients. The employee patient has a responsibility to the physician to provide him with complete and accurate data of his work place, as well as the reporting of all his symptoms and the factors relating to the illness. In short, everytime a working man goes into a doctor's office when he is being treated for an illness, he should provide the doctor with some information about his occupation, the number of years he was employed at that type of work, the materials he handles in his work, and the substances to which he is exposed. It is because this information is seldom given to the doctor that difficulties have occurred and occupational diseases pass unrecognized. The patient should bring his work-place to the doctor and the doctor should visit the work-place to study the occupational exposures and become acquainted with the hazards involved. In this latter regard, frequently there are changes in the mixture of substances sold under trade names with which the manager or safety director of a plant may be unacquainted. Or, because of some new process of manufacture, the final product sold may not be just one substance, for example not gasoline but gasoline-benzene, capable of producing typical symptoms of benzene poisoning.

The second point relates to the frequent desire in compensation cases to have a very narrow requirement that the symptoms conform exactly as to what is quoted in the text books. This is unwise because a wide range occurs in the symptoms of the nonindustrial diseases which all the physicians recognize as variations which can occur in different persons and under different physiological conditions. Similarly, variations can occur in the symptoms of occupational diseases. Instances have been reported of how one individual in exposure to the same substances can be markedly affected in a shorter period of time while others do not appear affected at all; Under the same conditions of exposure to a toxic substance, certain common symptoms will occur, but then there will be other symptoms observed in some employees and not in others. Although the so-called classical symptoms of occupational diseases do occur, one should not require each occupational disease to have all the symptoms that have been ascribed to that particular toxic substance.

The physician in interpreting these symptoms should be cautioned in accepting without reservation the results of animal experiments in the field of industrial poisons.³ Although there are consistent observations in animals and humans, marked differences have been found for some substances and this possibility always exists. Furthermore, such animal experiments are usually conducted at room temperatures. Under such

³ HAMILTON & HARDY, *INDUSTRIAL TOXICOLOGY* (2nd ed. 1949).

conditions it may be possible that the particular solvent may be rated as harmless in ordinary use; however, in industry the solvents may be subjected to heat in certain processes and the heat increases their toxicity. We will take as an example, methyl cellosolve, which is used as a solvent for the stiffening material on shirt collars. When the fabric is pressed with a hot iron, the solvent volatilizes and severe poisoning in a shirt factory has been reported.⁴ It must be remembered in correlating symptoms with exposure that heat and heavy work usually increase both the rate and depth of respiration and so hasten the absorption of the poison.

In essence, then, the attorney is dependent upon the physician for the decision of whether an illness is occupational or not. The physician has the responsibility of identifying these symptoms and their correlation with all the factors of the working environment, their variation in work processes and the chemical form and concentration of the toxic dusts, fumes, vapors, mists or gases, etc. There are a limited number of biological tests that provide objective measures of absorption. More frequently, as in the case of organic substances, there are presently no means of identifying the complex substance as metabolites in the body tissue or fluids. There must be reliance, then, to a certain extent on the frequency of association of illness and the specific exposure, recognizing again the exceptions and differences in observations which can occur as I have referred to previously. The diagnosis of an occupational disease requires a more thorough investigation and correlation than a nonoccupational disease for the latter does not require consideration of the toxic substances in the working environment. Lung biopsies, for example, may be necessary to differentiate diffuse pulmonary lesions and to establish the diagnosis of an occupational pneumoconiosis. Because of these various factors, I do believe that provisions should be made within the Workmen's Compensation Act for payments to the physicians for those tests that are conducted to determine whether a disease is occupational or not, even if the disease is finally found to be nonoccupational. At present, the law only provides payment when the disease is established to be occupational in origin.

STATUTES OF LIMITATIONS

Occupational Disease

The requirements that an occupational disease claim or death notice must be filed within six months after disability began and that exposure must have occurred within twelve months of disability is not consistent with our present-day knowledge of occupational diseases.⁵ There is adequate documentation in the scientific literature on occupational diseases to question the validity of the present Ohio statute of limitations. For example, in reference to literature concerning benzol in which the disease

⁴ Donley, *Toxic encephalopathy and volatile solvents*, 18 J. INDUST. HYG. & TOXICOL. 571 (1936).

⁵ OHIO REV. CODE §§ 4123.68 (Baldwin Supp. 1958), 4123.85 (1953).

or illness occurred after twelve months following cessation of exposure, it was found that injury to the bone marrow, once started, progresses without any further intoxication. In Hunter's cases⁶ (two—both fatal) they had no exposure to benzol for sixteen or seventeen months prior to treatment. Smith⁷ reported a case of benzol poisoning of a rotogravure printer who died of aplastic anemia, four years after exposure ceased. Meyer⁸ reported a case of extreme aplasia of the bone marrow, nine years after exposure to benzene ceased. Other cases, undoubtedly, have occurred of a similar nature. Not only aplastic anemia but leukemia may occur under similar circumstances. Rachner⁹ reported a case described as "chloroleukemia" in which exposure to benzol had ceased over two years prior to the development of the illness. A case of benzol poisoning in Ohio,¹⁰ after an interval of three years away from exposure, has been reported to me. This individual had thirty-six blood transfusions. He died six days after his claim was denied.

In another example, reference is made to a group of diseases, the occupational cancers, in which the cancer develops many years after exposure ceases. This observation is now common knowledge. Skin cancer from pitch tar or tar products and paraffin is known to occur fifteen or more years after exposure. A man may have changed jobs a number of times since the initial exposure to the carcinogenic agent. Still another illustration relates to the inhalation of dust in the manufacture of chromates producing cancer of the lung from this type of exposure as reported by Machle¹¹ and Mancuso¹² showing the latent period of the cancer to range from a few years to over twenty years. In the case of bladder cancer due to exposure in the manufacture of beta naphthylamine, a dye, which may occur twenty or more years after exposure, there is adequate scientific documentation showing a cause and effect relation in experiments with animals as well as the actual observation of workers exposed in the dye industries in this country and abroad.¹³ I might add that in the case of

⁶ Hunter, F.T., *Chronic exposure to benzene (benzol)-II, The clinical effects*, 21 J. INDUST. HYG. & TOXICOL. 331 (1939).

⁷ Greenburg, Mayers, Goldwater & Smith, A.R., *Benzene (Benzol) poisoning in the rotogravure printing industry in New York City*, 21 J. INDUST. HYG. & TOXICOL. 395 (1939).

⁸ Meyer & Guisberg, *Aplastic anemia*, 24 J. INDUST. HYG. & TOXICOL. 37 (1942).

⁹ Rachner, *Chloroleukemia as a result of benzol poisoning*, 27 ABSTR. IN J. INDUST. HYG. & TOXICOL. 93 (1945).

¹⁰ May 1958 (Personal Communication).

¹¹ Machle, Willard & Gregorius, *Cancer of the respiratory system in the United States chromate-producing industry*. 63 PUBLIC HEALTH REPORTS No. 35 (August 27, 1948).

¹² Mancuso, *Occupational cancer and other health hazards in a chromate plant; A medical appraisal-I. Lung cancer in chromate workers*, 20 IND. MED. & SURGERY 358-363 (August 1951).

¹³ Hueper, *Recent developments in environmental cancer*, 58 AMA ARCHIVES OF PATHOLOGY, 475-523 (November 1954).

carcinogenic agents, the organs in direct contact with the chemical may not necessarily be the ones to be affected, for example, the absorption of beta naphthylamine in the skin and by inhalation of the lungs produces tumors in the bladder and kidney where the metabolites are excreted.

Any thorough review of the occupational disease reports in the scientific literature will provide additional illustrations of other chemicals in which the development of disease or illness occurs long after exposure ceased, beyond the twelve months limitation statute.

I should like to direct attention to the other aspects of the statute of limitations, the requirement that the occupational disease claim be filed within six months after disability begins. The medical aspects of this requirement are important. The way the law operates, an employee may become sick, totally disabled and quit work because of an illness or disease that neither he nor his physician recognized as being occupational in origin. Then after six months, still ill, he continues to seek medical consultation. The specialist in occupational medicine then diagnoses the case as one that has been and is an occupational disease. Technically the employee is disqualified from filing a claim because the law specifies "within six months of disability," rather than six months of diagnosis. Why is the employee not given the right to file his claim within a period of six months after diagnosis by a licensed physician similar to the right given in the case of silicosis and the dust disease claims?

Silicosis

The statute of limitations provides that exposure to silica dust must have occurred within eight years of disability or death. Let us illustrate this point by an actual case. Here we have a man presently working as a janitor for five years, with admittedly no silica dust exposure on the job, but he is totally disabled, medically and factually from silicosis as of a given date, and he files a claim in a period of time provided by law within one year after his disability begins. The silicosis claim is disallowed because there was no injurious exposure within the last eight years. This case illustrates, medically, that a person may have a total disability due to silicosis and be denied compensation because exposure did not occur within the eight year period.

This legal provision, in general, is not consistent with the knowledge relative to variations in latent periods of occupational diseases due to different substances as related previously and the intervals between diagnosis and total disability. The interval from first diagnosis of silicosis to permanent disability can range from ten to twenty years or more. It has been established that lung fibrosis can and does continue in an individual who has developed silicosis and ceased exposure. This subsequent aggravation of disability is certainly related to the occupational exposure and is a result of continued physiological changes which occur from the presence of the already inhaled and permanently fixed silica in the lung tissues and the associated changes which occur in the cardio-pulmonary system.

Berylliosis

The Ohio statute of limitations provides that injurious exposure must have occurred within eight years of disability or death.¹⁴ There is now ample scientific data to show that lung and systemic changes due to berylliosis have occurred long after cessation of exposure, beyond the time limitation of eight years. The symposium¹⁵ on beryllium at the Massachusetts Institute of Technology revealed that cases had developed from ten to fifteen years after exposure ceased. These examples point to the necessary problem of qualifying occupational diseases for compensation when they become recognized through scientific studies and investigations, with a much broader interpretation of time limitation requirements.

The realization also exists that there is not sufficient application by the general practitioner or physician of some of the technical knowledge about occupational diseases which has already been accepted and recognized in the specialized literature. One wonders, for instance, about those individuals who have left an industry because of an illness which was not recognized as an occupational disease by themselves or the physician. These workers have carried the expenses of the medical bills for treatment and diagnosis for what they thought was a nonoccupational disease. Then, later, after twelve months away from the ceased exposure, they learn from the medical reports or a health department study that their illnesses were actually occupational in origin. Or, look at the case of the deprived family who subsequently learn that the death of their breadwinner some years ago was really due to chemicals in the working environment. Just what recourse is there for families and workers in the consideration of occupational diseases to allow compensation for such just claims?

LUNG DISEASES DUE TO DUST

Why has it been that in the entire Workmen's Compensation Act, the act will compensate a person totally disabled—if the person is totally disabled, and compensate for partial disability—if partial disability has occurred, or pay just medical expenses, and will pay any one of these three types or all three, but will not do this in the case of silicosis or any other lung disease due to dust? There, the individual must be totally disabled—the law says so, but why? Why has the act earmarked and set apart only one disability—total disability—for one type of disease, silicosis, and the other lung diseases due to dust? Why is silicosis singled out and why require all the other dust lung diseases (exclusive of berylliosis) to be confined to all the conditions, restrictions and limitations of the act relating to silicosis?

Silicosis

According to the provisions of the act, in the case of silicosis an in-

¹⁴ OHIO REV. CODE § 4123.68(V), (W) (Baldwin Supp. 1958).

¹⁵ Harriet Hardy, Massachusetts General Hospital, Boston, Mass. (Personal Communication, June 1958).

dividual with demonstrable lung disease and moderate or severe disability is not entitled to medical expenses and the case is disallowed unless he is totally disabled. This applies to the other chest lung diseases as well. Yet, in any other comparable injury or disability that is demonstrable, medical bills are paid even if the individual is not totally disabled (beryllium does not have this stipulation). This statement is made notwithstanding the provisions of change of occupation for persons with silicosis.

Silicosis as a disease has probably had more references in the medical literature and been more readily recognized as an "occupational" disease than any other industrial disease. There is no need, therefore, to provide an extensive discussion of this disease but rather to limit ourselves to those provisions of the act which warrant reconsideration as far as the medical aspects of the Workmen's Compensation Act is concerned. The basic point is that medically there is no disagreement but rather wide-spread recognition that in silicosis extensive fibrosis occurs, demonstrable by x-ray and by autopsy, that infection may occur, that secondary effects on the heart (cor pulmonale), because of the impediment to the flow of blood through the lung can occur and is not unusual, and that various stages of disability do occur. Marked as is the degree of pulmonary disability which does occur, yet, because of the legal provision of total disability requirement, no compensation is allowed. This represents one side of the problem but even more striking is the medical recognition that these persons with silicosis, before total disability is diagnosed, are and have been markedly ill requiring frequent medical treatment and prolonged care. Even so, although this is an occupational disease, their medical bills cannot be paid until total disability has been recognized by the Silicosis Board of Referees. This places the intervening burden of a prolonged illness and financial costs on the silicotic and his family.

The definition of silicosis as specified by the act states: "Silicosis means a disease of the lungs caused by breathing silica dust (silicon dioxide) producing fibrosis nodules distributed through the lungs and demonstrated by x-ray examination or by autopsy."¹⁶ Under the terms of the act, silicosis to be recognized for compensation purposes must have reached the stage of total disability and be due specifically to silicon dioxide. In other words, the present provision of the act would exclude silicates and other dusts, other than silicon dioxide.

Silica or silicon dioxide is the combination of one silicon atom with two atoms of oxygen which gives it a specific formula of SiO_2 . Free silica means that this specific compound is present chemically uncombined with any other element or groups of elements such as is the case with the silicates. The most common form of free silica is quartz which is found in minerals and clays. Other forms of free silica are tridymite, cristobalite, lechatelierite and amorphous silica. These silicas all have the same chemical make-up (silicon dioxide) but vary in their physical forms. An example of

¹⁶ OHIO REV. CODE § 4123.68(W) (Baldwin Supp. 1958).

this is when quartz is heated to high temperatures, it is changed into cristobalite and tridymite. The only change involved was a change in the crystal-line structure from one type of crystal to another type of crystal with no change in the chemical make-up. The amorphous silica is silicon dioxide with no determinable form or crystalline structure.

There is increasing evidence that the onset of silicosis can be hastened by the inhalation of other substances along with silica. The alkalinity associated with sand in a scouring preparation was reported¹⁷ to have caused silicosis in from 20 to 26 months ("galloping" silicosis) in a small group of exposed individuals; the silica dust concentration was tremendously high as far as can be gathered from the original report. Rapidly progressive silicosis has been previously reported in this country¹⁸ from the same cause. Talcosis of unusually rapid development (16 to 24 months) was reported¹⁹ also under circumstances of excessively high talc concentrations. Flouride in the form of fluorspar, in mixture with quartz, has also been reported²⁰ experimentally to induce more intense fibrosis than quartz alone although the latter may be present in only minute amounts (1 per cent) in the mixture.²¹

Frequently the question is asked what is an injurious exposure to silica dust? The interpretation of "injurious" should not be based upon a fixed arbitrary level or concentration of silica in the atmosphere. Often reference has been made to some regulations which have been adopted by governmental agencies in this state or other states and a claim disallowed, because the concentration found at the plant, at the time of investigation, did not reach the level as specified in such regulations. As expressed earlier in this paper, the dust counts at the time of investigation and the percentage of free silica, etc. may have no bearing whatsoever as to the true exposure several years, or five or ten years, previously. Actually, the concentration of substances varies within a shift, day by day, according to workers, production schedules, and changes in ventilation installations. The word "injurious" signifies the accumulative effect of the total quantity of exposure that the individual was exposed to over a period of time. Further, an exposure to silica of a certain quantity or concentration which may not be injurious to a person without the disease may be injurious to one who

¹⁷ Desoille, Tara, Delplace, & Cavigneaux, 14 *Arch. maladies profes. med. travail et securite sociale* 279-283 (1953).

¹⁸ Ritterhoff, 43 *AM. REV. TUBERC.* 117-131 (1941).

¹⁹ Alivisatos, Pontikakis & Terzis, 12 *BRIT. J. IND. MED.* 43-49 (1955).

²⁰ Policard & Collet, 14 *Arch. maladies profess. med. travail et securite sociale* 117-122 (1953).

²¹ Stokinger, *Toxicologic Aspects of Occupational Hazards*, 7 *ANN. REV. MEDICINE* 179 (1956). References 17, 18, 19, 20, & 21, *supra*, are from Dr. Stokinger's article.

already has silicosis. There is a marked variation in susceptibility of individuals. Cases have been observed of some persons exposed to small amounts of silica who have developed more disability than other persons who have been exposed to greater amounts of silica.

To answer this question, then, as to what is an injurious exposure to silica dust, I believe that an injurious exposure is any combination of the agent (toxic dusts, fumes, etc.), the environment (exposure conditions and duration) and the individual, that produces the disease. The presence of a specific disease, a disease already established and medically recognized, as due to a specific substance such as in silicosis, is evidence in itself that injurious exposure has occurred. The disease means and represents that the injury has actually occurred as has been demonstrated by physical and x-ray examination. In addition, any person with silicosis should not be further exposed to silica dust.

In the case of trauma, if an injury has occurred, then there must have been a causative agent. In this sense occupational lung disease due to dust is essentially slow trauma. If a man is hit by an automobile, the law is not concerned primarily with the intensity of the auto impact, but rather the result, the injury that occurred. The law can set a maximum level of speed or concentration of silica in an attempt to minimize injury, but neither a speed limit nor a predetermined concentration of silica can be considered an arbitrary level below which injury is not permitted to occur. The fact that any level of speed or concentration can be injurious is not, however, an argument that no controls should be instituted.

Asbestosis

Various stages of fibrosis do occur in other occupational lung diseases which require medical care, diagnosis and treatment. Asbestosis, for example, is a form of pneumoconiosis caused by long continued inhalation of asbestos dust. The chief symptoms usually associated with asbestosis are progressive dyspnea, cough hemoptysis, emaciation, weakness, poor chest expansion, curved finger nails or clubbed fingers, chest pain and cyanosis. The appearance of these symptoms can be explained on the basis of a more or less general fibrosis of the lungs and concomitant pathological changes such as emphysema, bronchieactis and fibrous pleurisy.²²

Despite these symptoms and the various stages of illness and disability which occur in asbestosis, medical payments for illness cannot be made nor can there be any recognition of partial disability according to the statutes. This is significant since

in well developed asbestosis the tissue alterations appear to be more widespread than in silicosis and even when the process is

²² DREESSEN & DALLA VALLE, A STUDY OF ASBESTOSIS IN THE ASBESTOS TEXTILE INDUSTRY, PUBLIC HEALTH SERVICE BULLETIN No. 241 (1938).

not severe in any one area, a major portion of the lung will be involved. Hence, there is relatively little truly entirely normal lung tissue in the person with clinically manifest asbestosis.²³

Alumina Abrasives

Another illustration relates to the lung changes which occur in the manufacture of alumina abrasives. Although the dust inhaled includes silica as well as alumina, this example demonstrates the extent of medical illnesses and complications which can occur requiring, of course, considerable medical attention and treatment over a prolonged period of time but for which no medical bills or partial disability can be recognized under the present statutes. The following statement is taken from the summary and remarks of the original report by Dr. C. G. Shaver:²⁴

A series of cases of lung diseases exhibiting what appear to be unusual features is described. These developed in connection with an industrial process which had previously been considered innocuous. The disease is essentially an industrial lung fibrosis, nonnodular in type. It may be rapidly progressive. Profound emphysema accompanies the invading fibrosis. Emphysematous bulbs and bullae occur on the visceral pleura. These are apt to rupture spontaneously and give rise to pneumothoraces. The etiology is doubtful. It is known that the process involves exposure to high concentrations of alumina and silica, both in a very fine state of division and to small quantities of many other substances.

Other lung diseases

Dr. Herbert E. Stokinger, Chief Toxicologist of the Public Health Service, recently reviewed the literature pertaining to dust diseases of the lung and the following abstracts from that review are pertinent to our discussion and are included for that purpose.

Isolated reports continue to appear with increasing frequency to incriminate many dusts, formerly considered inert, in the production of pneumoconiosis, such as sepiolite²⁵, corundum²⁶,

²³ Wright, George W., *Functional abnormalities of industrial fibrosis*, 11 AMA ARCHIVES OF INDUSTRIAL HEALTH No. 3, 196-204 (March, 1955).

²⁴ Shaver & Ridell, *Lung changes associated with the manufacture of alumina abrasives*, 29 J. IND. HYG. & TOXICOL. No. 3 (May, 1947).

²⁵ Parada, 2 MED. SEGUR. TRAB. 11-14 (1954).

²⁶ Hagen, 5 Z. ges. inn. Med. u. ihre Grenzgebiete, 31-34 (1950); Gartner, 6 ARCH. IND. HYG. & OCCUPATIONAL MED. 339-343 (1952).

feldspar²⁷, graphite²⁸, porcelain²⁹, barite³⁰, cement³¹, mica³², slate³³, kaolin³⁴, and a substance to which gas workers are exposed³⁵. Several reports in the last few years from the U.S. and England³⁶ are in agreement that coal miners' pneumoconiosis is an entity distinct from silicosis.

Pulmonary disability from the inhalation of grain dust is marked by dyspnea, chronic bronchitis, recurrent bronchial obstruction leading to clinically apparent emphysema, and was reported to occur among those working with seeds and grains for 10 or more years.

The capacity of organic as well as inorganic dust to produce lung changes has been documented in several reports. p-Dichlorobenzene was reported to have produced pulmonary granulomatosis in a middle-aged woman. Examination of excised lung tissue by polarized light revealed crystals believed to be p-DCB. The woman had been using p-DCB profusely for from 12 to 15 years on upholstery, carpets, and in clothes cupboards as an insecticide. Caution has been voiced on the inhalation of resin dust by Child & Clancy; during grinding and powdering of the synthetic resin, dryness of the mouth and nasal mucosa, coughing and sneezing, and other signs of respiratory irritation were in evidence. Experiments in animals produced wheezing, rales, blocking of major bronchi with distended resin granules, and salivation and atelectasis; the most prominent effects were caused from the loosely cross-linked resins. Dust and powder of Teflon (tetrafluorethylene polymer) have been known for several years to produce a respiratory condition akin to metal fume fever; more recent investigation of the inhalation hazards of heated Teflon showed

²⁷ Rotter & Gartner, 4 *Zentr. Arbeitsmed. u. Arbeitsschutz* 35-40 (1954).

²⁸ Boevt, 15 *Schweiz. z. allgem. Pathol. u. Bakteriologie* 548-552 (1953).

²⁹ Kirch, 25 *Beitr. Silikose-Forsch* 1-29 (1953).

³⁰ Pendergrass & Greening, 7 *ARCH. IND. HYG. & OCCUPATIONAL MED.*, 43-48 (1953).

³¹ Manciola, 23 *Rass. med. ind.* 7-16 (1954).

³² Ramaswamy, Venkatesh & Rama Rao, 35 *J. INDIAN INST. SCI.*, § A, 319-331 (1953).

³³ D'Onofrio, 21 *Rass. med. ind.* 344-346 (1952).

³⁴ Lynch & McIver, 30 *AM. J. PATHOL.* 1117-1127 (1954); Policard & Collet, 17 *Schweiz. Z. allgem. Pathol. u. Bakteriologie*, 320-325 (1954).

³⁵ Tyler, Gregory & Carson, 3 *TRANS. ASSOC. INDIAN MED. OFFICERS*, 246-249 (1953).

³⁶ Martin, 44 *AM. J. PUBLIC HEALTH* 581-591 (1954); Heppleston, 66 *J. PATHOL. BACTERIOL.* 235-241 (1953); Heppleston, 67 *J. PATHOL. BACTERIOL.* 51-55 (1954); Flinn, Seifert, Brinton, Jones & Franks, 270 *PUBLIC HEALTH BULLETIN (U.S.)* 118 (1941).

the evolution of highly toxic fluorine-containing decomposition products.

This all-too-brief review of lung diseases from inhaled particulates makes it difficult to escape the conclusion that all dusts, irrespective of their nature, if breathed in sufficient quantity and for sufficient time, may cause profound damage to the lung, and emphasizes the desirability of the physician's obtaining an accurate and thorough occupational history on individuals suspected of pulmonary disease.³⁷

These abstracts of the literature indicate that significant respiratory illnesses can and do occur due to the inhalation of dusts and, yet, under the provisions of the act, the medical bills for diagnosis and/or treatment technically cannot be allowed, unless total disability occurs. This requirement of the act, therefore, is not consistent with the provision cited under the act for injuries and other diseases, and it appears silicosis, in particular, and the dust lung diseases, in general, have been singled out with restrictive legislation.

Considerable progress has been made in the measurement of pulmonary disability since the act's first provisions relative to silicosis were specified. Although further refinements are desired, nevertheless, it is readily possible to delineate relative ranges of disability sufficient to establish when partial disability has occurred in cases of silicosis and other lung diseases.

The present statutes require that all respiratory diseases due to dusts be referred to the Silicosis Board of Referees for review and decision relative to diagnosis and disability.³⁸ The broad range of industrial dusts involved would indicate that there should be reconsideration of this provision of the Act.

Pulmonary Emphysema

There has been considerable discussion among the medical and legal professions concerning the word "emphysema." Medically speaking, emphysema is an over distention of the lung. There are several varieties. Emphysema can occur with or without chronic obstructive bronchitis. In the disabling emphysema, there is a loss of elasticity and obstruction to air flow resulting in impaired pulmonary function and dyspnea. There are exacting means of measuring emphysema and the severity of impairment by pulmonary function tests; these include the "timed" vital capacity, the maximal breathing capacity, measurements of residual volume and functional residual capacity, as well as distribution of air within the lung, and diffusion capacity studies. Some medical researchers feel

³⁷ Stokinger, *op. cit. supra* note 21, at 180-82. References 25 thru 36 are from Dr. Stokinger's article, but have been renumbered. Other references appearing in this excerpt have been omitted.

³⁸ OHIO REV. CODE § 4123.68(W) (Baldwin Supp. 1958).

that chronic irritant factors cause bronchial infection and obstruction which in turn induces emphysema. McLean³⁹ and others suggest that bronchiolitis is a main cause of emphysema.

In some areas of the country, emphysema is interpreted as a principal disability factor when it occurs with lung diseases due to dusty trades and it is considered compensable. In other areas, the presence of emphysema is interpreted as being due to some other cause and not due to the lung changes which produce the fibrotic process. In such instances the interpretation of silicosis cases with emphysema are labeled as noncompensable because of the presence of emphysema. At the present time a fair statement regarding the medical aspects of disability relating to emphysema would be this: (1) that although one cannot at present conclusively prove that emphysema is of occupational origin when it is associated with an occupational lung disease, one cannot equally and conclusively disprove that it is not; (2) that on this basis one could question the validity of the exclusion from compensation of a case of silicosis because of concomitant emphysema. Before this question can be settled medically, we need to know the etiology and the exact incidence of emphysema in the total population according to certain characteristics: age, sex, color, etc. We need, also, to know what the incidence of emphysema is in certain dusty occupations, foundry workers, miners, etc.

In reference to the question of disability, Dr. Hardy,⁴⁰ Chief of the Occupational Medical Clinic at the Massachusetts General Hospital, wrote recently to me of her observations which indicate the lack of correlation between chest x-ray picture, lung function studies and the pathology.

We have been studying since 1949 at the Massachusetts General Hospital, with support from the U. S. Public Health Service, job-related diseases of the chest. We have studied cases of asbestosis, chronic beryllium disease and, more recently, pneumoconioses due to silica inhalation and due to inhalation of mixed dusts such as those encountered in underground mining.

We have been struck, in preparing some material for publication on the subject, by the lack of correlation between the disability of the patients, the chest x-ray picture, the lung function studies and the pathology. This is, of course, very important to those having to consider reasonable legislation for compensation of job-related illness. We are particularly struck by the measurable disability in men with long years of exposure to respirable mine dust without distinguishing chest x-ray abnormalities.

We are also impressed by the similarity between the chest x-ray patterns resulting from such things as the inhalation of toxic beryl-

³⁹ McLean, *The pathology of emphysema*, Aspen Conference on Research in Emphysema (June, 1958).

⁴⁰ Personal Communication (June 1958).

lium compounds and the x-ray pattern of miliary tuberculosis and that puzzling disease, sarcoidosis.

RADIATION HAZARDS

A great deal of publicity is being given to radiation and its effect on health but I should like to point out that there are many different kinds of radiation and that these may be divided into two categories. The type of radiation that is receiving so much attention today, the type that is associated with atomic bombs, nuclear reactors, x-ray machines, etc. is the type of radiation which we call *ionizing* radiation.

The *ionizing* radiations which include x-rays, alpha-, beta- and gamma rays and others are capable of penetrating matter and of causing atoms and molecules to break up into electrically charged particles called *ions*. It is through this ionizing effect that body cells are damaged when humans are exposed to *ionizing* radiation.

The other kinds of radiation with which we are all familiar but which do not ordinarily come to mind when the subject of radiation is discussed are the *non-ionizing* radiations such as ultra-violet, infra-red, visible light and radio waves. All of these are electromagnetic radiations as are x-rays and gamma rays but they are not as penetrating as the latter and they do not produce ionization when they interact with matter.

The non-ionizing radiation, while not as newsworthy in these times as the ionizing radiations, are, nevertheless, capable of producing biological damage. We are all familiar with the painful and oftentimes serious burns received from exposure to ultra-violet radiation from the sun or from a sun lamp and any arc welder can tell you that viewing the flash of the electric arc without protection for the eyes will result, some four to six hours later, in a very painful feeling as if the eyes had been filled with sand. The ultra-violet radiation from some forms of electric arc welding is also capable of producing ozone and nitrogen oxides in concentrations sufficient to cause chemical pneumonia.

Likewise, infra-red radiation, the type of radiation emitted by a heat lamp or any heated object, is reported to have caused cataracts in the eyes of workers, such as glass blowers, whose eyes are continually exposed to infra-red radiation. And microwaves, which are the ultra-short radio waves used in radar and in the new electronic ovens, are also recognized as being capable of inflicting damage to the body if one is exposed to an intense source from too short a distance.

All of these non-ionizing radiations as well as the ionizing radiations have found application in industry and commerce and newer applications, particularly in the field of missiles and national defense, are being continually developed.

Since, in the popular sense, the term radiation has been used as if it were synonymous with *ionizing* radiation, only the ionizing radiations will be discussed in the remainder of this section. It is my feeling that

in certain respects the importance of radiation as a causative agent in occupational disease has, through widespread publicity, been over-emphasized to the point where it has become difficult for the layman to view the problem in its proper perspective.

Occupational illness due to radiation exposure has, of course, occurred and the potential hazard is increasing with the increased use of radioactive materials and with the utilization of nuclear energy. Yet the number of workers potentially exposed to radiation in Ohio represents only a minute fraction of the total industrial population and will remain so, even with the increased uses of radiation and nuclear energy. On the other hand, literally hundreds of thousands of workers are potentially exposed every day to toxic dusts, fumes, mists, vapors or gases. Many of these substances can produce occupational diseases every bit as serious and in many cases even more so than the illnesses produced by overexposure to radiation. But, because of the intense publicity, workers are much more frightened of radiation than they are of exposure to lead dust and fumes, in spite of the fact that there are probably more cases of lead poisoning in Ohio in one year than there are of radiation injury in the entire country in several years.

The use of x-rays in industry for the nondestructive testing of metal castings and welded metal products has been practiced for years, although a survey conducted by the Ohio Department of Health in 1950 showed that there were only sixty-nine industrial x-ray units in use in the state. An average of only two or three persons were potentially exposed to x-rays from each of these units. Possibly one or two dozen plants were using radium for the same purposes at that time. An additional fifty-six x-ray units, including x-ray diffraction units, electron microscopes, fluoroscopes, etc. were being used in college, university and industrial laboratories.

Industrial x-ray units ranged in energy from 100,000 volt units used for inspecting light aluminum castings to one and two million volt units used for x-raying heavy steel pressure vessels such as industrial boilers. Although it is quite possible that some workers may have been exposed to an excessive amount of radiation from such units, we, nevertheless, do not know of a bona-fide case of radiation injury resulting from the industrial use of x-rays in Ohio.

In contrast, thousands of gallons of carbon tetrachloride and other chlorinated hydrocarbon solvents are used each year in Ohio and many workers have suffered liver damage and other serious biological injury from overexposure to these solvents. Benzol, another industrial solvent, has produced serious blood diseases in workers; diseases every bit as serious as the blood changes that can be wrought by exposure to radiation.

Radioactive isotopes produced as by-products of nuclear reactors were made available to industry for peacetime applications in 1946 and by 1955 around 130 Ohio industries were using these radioactive materials. Radioactive cobalt was being used in place of x-rays and radium

to inspect castings and welds. Another very important use of these new radioactive materials was in the radiation thickness gage, a device that permits continuous, accurate measurement of the thickness of a sheet of material such as paper, rubber or even steel as it travels at high speeds over rollers in the manufacturing plants.

Even though the amount of radioactivity licensed by the Atomic Energy Commission for use in Ohio industry in 1955 was almost twice the entire world-wide supply of radium, the majority of the plants were using only a few milligrams or micrograms as tracers in research experiments.

The uses of these radioactive materials are expanding and nuclear reactors, which contain enormous quantities of radioactivity, are being built and operated in Ohio. But at the same time, hundreds of new chemicals, many of them of very high toxicity, are being introduced into industry. And, for some reason, probably because radiation is still a mysterious phenomenon to both labor and management, a danger that cannot be detected by the human senses, radiation in industry is almost always used with elaborate safeguards while an equally hazardous operation involving a toxic chemical may go on unnoticed a few yards away.

I do not wish to convey the impression that I am underestimating the potential threat of radiation as a causative agent in occupational disease. It is a threat that deserves serious consideration and attention. But, I do feel that it should be considered as one of a large number of causes of occupational diseases, not to be singled out as a single overwhelming threat to the health of a large number of workers. I am sure that for many years to come, the number of occupational diseases resulting from exposure to radiation will constitute only a minute fraction of the total number of occupational diseases barring some unforeseen catastrophic accident such as major nuclear reactor excursion.

REFERENCE DATA ON SPECIFIC SUBSTANCES

Appendix I includes data on fourteen specific chemicals and their compounds relating to the occupations involved and the symptoms of industrial poisonings. This material provides only a small indication of the magnitude of the occupational disease exposures in which toxic substances are involved in the industries of Ohio. Similar data may be obtained from a variety of sources.⁴¹

Frequently physicians and attorneys desire some reference points in regard to the range or levels at which exposure to certain substances might

⁴¹ GLEASON, GOSLIN & HODGE, CLINICAL TOXICOLOGY OF COMMERCIAL PRODUCTS (1957); HAMILTON & HARDY, *op. cit. supra* note 3; HENDERSON & HAGGARD, NOXIOUS GASES (2nd ed. 1943); HUEFER, OCCUPATIONAL TUMORS & ALLIED DISEASES (1942); SAX, *op. cit. supra* note 1; SCHWARTZ, TULIPAN & BIRMINGHAM, A TEXTBOOK OF OCCUPATIONAL DISEASES OF THE SKIN (1958); VON OETTINGEN, THE HALOGENATED HYDROCARBONS-TOXICITY AND POTENTIAL DANGERS, U.S. DEP'T. OF H.E.&W., PUBLIC HEALTH SERVICE PUB. NO. 414 (1955).

be related to occupational diseases. We have not attempted to provide any such fixed levels of toxic substances but rather have included instead, in Appendix II, the Report of the Committee on Threshold Limit Values of the American Conference of Governmental Industrial Hygienists to show how complex the problem is and to indicate the many substances that are already identified in occupational exposures. The list does not include other substances used in industry or combinations of substances for which there is no toxicological data. The second purpose is to emphasize the high level of laboratory and engineering services required in the sampling and identification and analyses of these substances in industry by official and private agencies evaluating an occupational disease problem or providing data on an occupational disease claim.

In closing I should like to emphasize that although modification of the Workmen's Compensation Act should be considered, there is a basic need for a well coordinated program of prevention by the three state agencies who have responsibility in the field of industrial health: the Department of Workmen's Compensation, the Department of Industrial Relations, and the Ohio Department of Health. The objective is the prevention and control of occupational diseases in Ohio.

APPENDIX I

The following articles prepared by Dr. Mancuso which appeared in the OHIO STATE MEDICAL JOURNAL are illustrative: *Antimony and Its Compounds* (Sept. 1955); *Cadmium and its Compounds* (Oct. 1955); *Carbon Disulfide* (Jan. 1955); *Carbon Tetrachloride* (Feb. 1955); *Chlorine and Hydrochloric Acid* (Nov. 1955); *Cyanides* (Dec. 1955); *Hydrogen Sulfide* (June 1955); *Manganese* (March 1955); *Mercury and Its Compounds* (April 1955); *Methyl Alcohol* (May 1955); *Phenol and Related Compounds* (July 1955); *Zinc and Its Compounds* (August 1955); *Lead and Its Compounds*, (prepared for Ohio Dept. of Health) A NOTE ABOUT OCCUPATIONAL DISEASES—ESPECIALLY LEAD POISONING (1958); *Benzol (abstract)* (data from the Ohio Dept. Health). The occupations recorded below are taken from the industrial survey of the state of Ohio by the Ohio Department of Health in 1940. No similar survey has been conducted since this time.

INDUSTRIAL POISONING

ANTIMONY AND ITS COMPOUNDS

OCCUPATIONS:

Occupations in Ohio where contact with antimony and its compounds are indicated are listed as follows:

Assemblers (chemicals; metal furniture; storage batteries)	Beaders (tin and enameled ware)
Babbiting machine operators (brass factories)	Buffers (other metals)
Babbit pourers (foundries)	Burnishers (rifle barrels)
Banbury operators (rubber tires)	Calico printers
Band saw operators (brass factories)	Casters (other metals; storage batteries)
	Chargers (zinc smelting)

Checkers (storage batteries)	Mill men (rubber tires)
Chemical mixers (toys and unclassified novelties)	Mixers (chemicals; match factories)
Color makers	Monotype operators (printing)
Compounders (rubber tires)	Mordanters
Copper refiners	Mould cleaners (storage batteries)
Die cast operators (brass factories)	Moulders (brass factories; electrical machinery; metal furniture)
Dippers (potteries; tin and enameled ware)	Multi-cut saw operators (brass factories)
Dye makers	Oilers (blast furnaces; storage batteries)
Electroplaters	Packers (chemicals; storage batteries)
Element burners (storage batteries)	Pasters (toys and unclassified novelties)
Enamel bakers (foundries)	Punch press operators (storage batteries)
Enamel makers (tin and enameled ware)	Receiving clerks (brass factories; rubber tires)
Filers	Refiners (storage batteries)
Foremen (chemicals; metal furniture; storage batteries)	Rubber (red) workers
Foundry men (chemicals; storage batteries)	Scalemen (storage batteries)
Fuse assemblers (chemicals)	Shakeout men (brass factories)
General truckers (storage batteries)	Shot makers
Glass mixers	Spray painters (foundries; tin and enameled ware)
Hand moulders (storage batteries)	Stampers (toys and unclassified novelties)
Hand tool operators (brass factories)	Stencil men (storage batteries)
Inspectors (storage batteries)	Storage men (chemicals)
Janitors (printing)	Strappers (storage batteries)
Laboratory assistants (storage batteries)	Strap supply men (storage batteries)
Lathe operators (other metals)	Supply men (storage batteries)
Lead smelters	Sweepers (storage batteries)
Linotype operators (printing)	Tool makers (storage batteries)
Loaders (storage batteries)	Touch up men (storage batteries)
Machine builders (storage batteries)	Truckers (storage batteries)
Machine operators (blast furnaces; storage batteries)	Unloaders (storage batteries; car and railroad shops)
Machinists (blast furnaces; shoes)	Vulcanizers
Mechanics (storage batteries)	Welders (foundries)
Melters (brass factories; printing)	Wheelers (storage batteries)
Meter assemblers (electrical machinery)	Zinc refiners

INDUSTRIAL HEALTH ASPECTS

SYMPTOMS OF INDUSTRIAL POISONING:

(Antimony) Symptoms of occupational antimony poisoning are difficult to define. Most of the antimony of industry contains traces of arsenic and it is practically always used in conjunction with lead. It is evident that under such conditions a clear clinical picture would be impossible. However, symptoms of antimony poisoning have been described as follows: tightness of the chest, cough, swelling of the throat, gastro-intestinal disturbances, pustular eruptions especially the scrotum, difficult urination,

loss of sexual desire, eosinophilia, and nervous symptoms of many different varieties.

(Antimony hydride) The toxic action of antimoniuiretted hydrogen or stibine is similar to arsine but less potent. It attacks the central nervous system and the blood. The symptoms of acute poisoning are headache, weakness, nausea, retarded breathing, weak, slow, and sometimes irregular pulse, lowered temperature, and diuresis. Antimoniuiretted hydrogen is often encountered as a by-product as in the preparation of hydrogen from zinc. Other toxic impurities such as arsine frequently appear in the same processes.

(Antimony trioxide) Pneumonitis has occurred among workers in an antimony smelter; extensive pneumonitis in animal exposures of guinea pigs and chronic lipid pneumonia in rats have been recorded. Pulmonary inflammation has been noted as a sequel to therapeutic intramuscular administration of antimony compounds.

(Antimony trisulfide) Brieger recently established occupational and pathological evidence that exposure to this compound has produced *heart disease*. Experiments were conducted on rats and rabbits which revealed definite and consistent changes in the EKG. T-waves were especially affected. Marked dilatation and very flabby myocardium was observed.

BENZOL

INDUSTRIES:

Ohio industries using benzol are as follows:

Aircraft	Other chemicals
Automobile factories	Other foods
Blast furnaces	Other textiles
Charcoal and coke	Paint and varnish factories
Chemicals	Paper box factories
Clock and watch factories	Paper and pulp mills
Cotton cloth	Patent medicine, drugs
Dental supplies	Petroleum refineries
Dry cleaning and dyeing	Pianos and organs
Dyestuffs, ink, etc.	Printing
Foundries	Rubber, auto accessories
Garages	Rubber tires
Instruments	Shoes
Laundries	Soap factories
Leather belts and goods	Suits, coats, and overalls
Metal furniture	Trunks and suitcases
	Wood, wicker, etc.

OCCUPATIONS:

Occupations in Ohio where benzol is used are:

Air bag builders (rubber tires)	Battery men (garages)
Air bagmen (rubber tires)	Bay makers (other rubber factories)
Assemblers (other rubber factories, rubber tires)	Bench workers (dental supplies, instruments)
Backers (shoes)	

- Benzol house operators (blast furnaces, charcoal and coke)
Bias cutters (rubber tires)
Book bindery workers (printing)
Bookers (rubber tires)
Buffers (rubber tires)
Builders (rubber tires)
Case makers (leather belts and goods)
Cement patchers (rubber, auto accessories)
Cementers (rubber tires, shoes, suits, coats, overalls)
Checking machine operators (rubber tires)
Chemists (blast furnaces, charcoal and coke, dyestuffs, ink)
Commercial artist (rubber)
Compo conveyors (shoes)
Compositors (printing)
Covering men (aircraft)
Creasers (leather belts and goods)
Cutters (rubber tires)
Delivery boys (printing)
Dippers (rubber), dusters (rubber tires)
Electro-brass platers (rubber)
Etchers (metal furniture)
Embossers (shoes)
Experimental men (rubber tires)
Eyelet stayers (shoes)
Fillers (rubber tires, shoes)
Filling machine operators (other chemicals)
Finishers (dry cleaning and dyeing, rubber tires, wood, wicker, etc.)
Fitters (shoes)
Flag sprayers (rubber tires)
Flap layers (shoes)
Foremen (other textiles, pianos and organs, rubber tires)
Furniture cleaners (dry cleaning and dyeing)
Hand make-up men (printing)
Insole men (shoes)
Laboratory workers (dental supplies)
Laborers (dental supplies, blast furnaces, dyestuffs, ink, etc., paint and varnish factories, shoes, rubber tires)
Leather skiving operators (shoes)
Leather workers (shoes)
Liners (shoes)
Lock-up men (printing)
Millmen (rubber tires)
Millwrights (other textiles)
Mixers (rubber tires)
Oiling off men (pianos and organs)
Operators (chemicals, petroleum refineries, shoes)
Painters (foundries, pianos and organs, rubber tires)
Plater pressmen (chemicals)
Ply benders (rubber tires)
Press feeders (instruments, printing)
Press men (printing, chemicals, other rubber factories)
Press room devils (printing)
Printers (metal furniture, other foods, paper and pulp mills, patent medicine and drugs, -rubber tires, soap factories)
Printer's helper (printing, paints and varnish)
Production men (rubber tires)
Pump men (garages)
Raw stock preparation men (other textiles)
Repairmen (rubber tires, shoes, trunks and suitcases, garages, clock and watch factories)
Resizers (rubber tires)
Rubbing men (pianos and organs)
Rug cleaners (dry cleaning and dyeing)
Sewers (leather belts and goods, shoes)
Shell coverers (rubber tires)
Soap mixers (petroleum refineries)
Sock liners (shoes)
Splicers (rubber tires)
Spotters (dry cleaning and dyeing, laundries)
Sprayers (pianos and organs, rubber tires)
Spreaders (cotton cloth, other textiles)
Stampers (rubber tires)
Stock boys (rubber tires)
Stock preparers (rubber tires)
Supervisors (rubber tires)
Tank cleaners (paint and varnish factories)
Tapers (rubber tires, shoes)
Testers (paint and varnish factories)
Thinners (paint and varnish factories)
Tire study men (rubber tires)
Tread washers (rubber tires)
Treers (shoes)

Trimmers (wood, wicker, etc.)	Utility men (other chemicals)
Truckers (rubber tires)	Valve stem treaters (rubber tires)
Tube curing men (rubber tires)	Vulcanizers
Type setters (instruments, rubber tires)	Wood heel coverers (shoes)
Upholstering men (aircraft)	Wrappers (other chemicals)
	Wrinkle pullers (other textiles)

INDUSTRIAL HEALTH ASPECTS

MODES OF ENTRANCE

Inhalation. Ingestion may also be considered, and some writers are of the opinion that in chronic cases absorption by the skin may be accepted.

SYMPTOMS OF INDUSTRIAL POISONING

Acute poisonings due to inhalation of the fumes cause symptoms referable to the central nervous system with tremors, salivation, violent twitchings, exhaustion, paralysis, increased respiratory rate followed by slow respiratory rate, rapid pulse and decreased temperature. Lethal amount causes narcosis and convulsions with death from paralysis of the respiratory center.

In chronic cases occur headache, malaise, weakness, somnolence, vertigo, cough, dyspnea. Fever and lesions in the mouth are present in severe cases. There are petechiae, and hemorrhages from the mucous membranes. Changes in the vessel walls lead to multiple hemorrhages into skin and subcutaneous tissues, gums, nose, stomach, intestine, and uterus; menorrhagia and metrorrhagia occur in females. In some cases hemorrhages are absent. Blood forming organs are stimulated then depressed with a marked agranulocytic anemia of aplastic type. Leukopenia is usually in excess of the anemia although the reverse may occur. The anemia may progress though the cause is removed. The Arneth Count shows a marked swing to the right with absence of myelocytes; myoblasts, and lymphoblasts; there is usually a decrease in platelets, slow coagulation of the blood, no retraction of the clot. A distinct and rapid decrease in the percentage of inorganic sulfates of the total sulfates in the urine is noted even before the symptoms occur. However, the inorganic sulfate test is useless when benzol is mixed with carbon tetrachloride since that substance causes an increase in organic sulfates in the urine.

Local action on the skin and mucous membrane is frequent. There may be a discomfort, dry sensation, swelling, erythema or eczema, especially on exposed portions of the body. Slight cases may recover and yet be associated with severe and prolonged sequelae. Individuals may become hypersensitive and display symptoms of acute intoxication from small doses of benzol which were readily tolerated at first.

ELIMINATION

The products of its oxidation, represented by phenol, pyrocatechol, hydroquinone are eliminated slowly by the urine as sulfates.

CADMIUM AND ITS COMPOUNDS

OCCUPATIONS:

Buffers (electroplating)
Cadmium-alloy makers
Cadmium and cadmium-compound makers
Cadmium platers
Cadmium-vapor-amp makers
Calico printers
Chargers (zinc smelting)
Color makers
Cupola tenders (foundries)
Electroplaters (automobile factories)
Electroplaters
Glass-blowers (glass factories)
Glass colorers
Grinders (electroplating)
Laborers (chemical, glass factories, electroplating, toys and unclassified novelties)
Lithopone makers
Operators (welding, forging and heat treating)

Painters (foundries)
Platers (foundries, welding, forging and heat treating, machine shops, brass factories electroplating, metal furniture, printing and publishing, electrical machinery)
Polishers (welding, forging and heat treating, brass factories, electroplating, other metals)
Process men (chemical)
Solderers
Solder makers
Storage-battery makers
Supervisors (welding, forging and heat treating)
Technical men (chemicals, electrical machinery)
Welders
Zinc smelters and refiners

INDUSTRIAL HEALTH ASPECTS

MODES OF ENTRANCE:

Industrial poisoning is usually caused by inhalation of dust or fumes; Nonindustrial poisoning, usually caused by ingestion of cadmium compounds.

INDUSTRIAL POISONING:

Symptoms usually follow absorption of fumes or dusts from the respiratory tract. There is usually one or more of these symptoms: Dryness of throat, headache, cough, weakness, anorexia, pain in the chest, moderate fever, inflammation of the respiratory system with bronchitis. In severe exposures, bronchial pneumonia, edema and atelectasis may follow.

Strikingly variable syndromes of chronic cadmium poisoning have been reported from battery factories, including a peculiar osseous lesion termed Milkman's syndrome; the latter starts with pain in the lumbar region and the lower limbs and with gait disturbances—the roentgenograms showing in addition to osteoporosis, bands which are less opaque and which have a course transverse to the longitudinal axis of the bones.

Dental alterations have also been described as signs of chronic cadmium poisoning, in the form of a golden-yellow discoloration of dental cement and neck; in view of the fact that dissolved cadmium may be precipitated in the form of yellow cadmium sulfide by hydrogen sulfide, this phenomenon may correspond to the formation of heavy metal bands of the gums. The dental phenomenon usually appears after two years

of work, but may appear within a few months, and is considered an indication to give up work with cadmium.

STORAGE:

Mainly in the liver, less in the bones and kidneys.

ELIMINATION:

Slowly via the kidneys and gastrointestinal tract.

PERTINENT NOTES:

Cyanogen, a poison similar to prussic acid, has been reported in connection with cadmium plating processes. *Arsine*, a poisonous gas, may be formed in the refining of cadmium metal.

CARBON DISULFIDE

OCCUPATIONS:

Acetylene workers	Insecticide makers
Ammonium-salts makers	Match-factory workers
Artificial-silk makers	Mill men (rubber)
Asphalt testers	Mixers (rubber)
Carbanilide makers	Oil extractors
Carbon-disulfide makers	Painters
Cellulose workers	Paint makers
Cementers (rubber shoes)	Paraffin workers
Cement mixers (rubber)	Putty makers
Driers (rubber)	Reclaimers (rubber)
Dry cleaners	Smokeless-powder makers
Electroplaters	Sulphur extractors
Enamelers	Tallow refiners
Enamel makers	Transparent-wrapping-material workers
Explosive workers	Vulcanizers
Glue workers	

INDUSTRIAL HEALTH ASPECTS

MODES OF ENTRANCE:

Inhalation or by contact with the skin.

SYMPTOMS OF

INDUSTRIAL POISONING:

Acute and chronic types are recognized. Symptoms of acute poisoning are characterized by a stage of well-being, excitement like alcoholic intoxication, hallucinations, sometimes uncontrollable laughter, headache, throbbing of temples, palpitation, fainting and drowsiness, irritability, and insomnia.

Digestive disturbances follow the foregoing symptoms; there is nausea, vomiting, loss of appetite, occasionally diarrhea, sometimes colic and constipation.

Carbon disulfide dissolves the lipoids of the blood and acts upon the

central nervous system and parenchymatous organs. There is weakness of legs, unsteady gait, incoordination, signs of fatigue, loss of memory and a mania with homicidal and suicidal tendencies reported.

Chronic symptoms may appear in a few weeks or months or after years of work. Following the exciting stage there may be a stage of depression, melancholia, faintness, giddiness, drowsiness, exhaustion, headache, drunken gait, and exaggerated reflexes. Usually a positive Romberg's sign is found. Peripheral neuritis, disturbances of digestion, taste, smell, and sight are present.

Progressive emaciation, atrophy of muscles, circulatory and respiratory troubles may continue. The patient cannot read because of disturbances of vision, color fields and retrobulbar neuritis. Recovery may be rapid or it may be slow and extend over several months.

Genital disturbances may include: in male, genital hyperaesthesia and exaggerated activity followed by impotence; in female, menstrual disturbances, ovarian pain, abortions and breast atrophy.

Locally, it has an irritant action; contact with the skin causes a sensation of burning and subsequently anesthesia, later dryness, erythema, eczema or pigmentation. Various cutaneous trophic changes have been described. Sweating may be abundant with the odor of carbon disulfide.

ELIMINATION:

Mainly through the pulmonary tract and in the urine, also through the sweat and intestinal tract. Elimination is slow, thus a cumulative action may be apparent.

CARBON TETRACHLORIDE

OCCUPATIONS:

Occupations in Ohio where contact with carbon tetrachloride was indicated are listed as follows:

Assemblers (foundries)	Mechanics (garages)
Blockers (hats)	Mill men (rubber tires)
Builders (rubber tires)	Operators (dry cleaning and dyeing)
Chemists (other chemicals)	Packers (printing)
Cleaners (foundries)	Paste makers (other chemicals)
Dental technicians (dental supplies)	Pharmacists (patent medicine, drugs)
Dry cleaners (laundries; dry cleaning and dyeing; suits, coats, and overalls; fur goods; hats; shirts, collars, and cuffs)	Plant patrol men (electrical machinery)
Firemen (other chemicals)	Pressers (dry cleaning and dyeing; suits, coats, and overalls)
Fillers (patent medicine, drugs)	Safety equipment men (storage batteries)
Finishers (dry cleaning and dyeing)	Seamstresses (dry cleaning and dyeing)
General porters (shirts, collars, and cuffs)	Shipping clerks (other chemicals)
Hatters (hats)	Sorters (dry cleaning and dyeing)
Inspectors (shirts, collars, and cuffs)	Spotters (dry cleaning and dyeing; laundries)
Labelers (patent medicine, drugs)	

Storage men (dry cleaning and dyeing)	Electroplaters
Superintendent (other chemicals)	Fire-extinguisher makers
Truck drivers (other chemicals)	Firemen
Utility men (other chemicals)	Lacquerers
Wash men (textile dyeing and finishing)	Metal-polish makers
Airplane dope workers	Paraffin workers
Cementers (rubber)	Perfume makers
Degreasers (textiles)	Rubber workers
	Vulcanizers

INDUSTRIAL HEALTH ASPECTS

MODES OF ENTRANCE:

Inhalation, ingestion and skin absorption.

SYMPTOMS OF INDUSTRIAL POISONING:

Loss of appetite, nausea, vomiting, cough, headache, somnolence, nervousness, marked excitement, mental confusion, vertigo, increased pulse and respiration, air hunger, weakness, burning sensation in the epigastrium, diarrhea, loss of weight, jaundice, secondary anemia, pain and tenderness over an enlarged liver, and tendency to intestinal hemorrhages.

Visual disturbances include blurred vision, color confusion and disturbance of near vision.

The urine shows increased acidity, increased phosphates, casts and albumin. There is suppression of urine, uremia, coma, convulsions, and death. At autopsy is found central necrosis and fatty degeneration of the liver and kidneys.

Fats, alcohol, and a negative calcium balance are stated to increase the susceptibility to carbon tetrachloride. There also occurs irritation of the eyes, nose, and throat, and of the skin. A mild addiction of this narcotic may take place.

McNulty states that those having nephritis, diabetes, myocardial degeneration, high blood pressure, or those using alcohol, should not work with carbon tetrachloride.

When the vapor of carbon tetrachloride contacts hot metal or an open flame, phosgene may be formed with severe irritant effect on the mucous membranes and respiratory tract.

ELIMINATION:

Mainly through the lungs, scarcely any in the urine.

CHLORINE AND HYDROCHLORIC ACID

OCCUPATIONS:

Acid polishers (glass factories)	Analysts (lime, cement, and artificial stone)
Acid treaters (foundries; glass factories)	Annealers (foundries)

- Assemblers (electric fixtures; electrical machinery; foundries; metal furniture)
Babbit room tenders (brass factories)
Battery men (garages)
Bead makers (rubber tires)
Bench workers (brass factories; dental supplies; foundries)
Bleachers (cotton cloth)
Body men (automobile factories)
Bottle sorters (soft beverages)
Brazers (other manufacturing plants)
Carbonizers (woolens and worsteds)
Chemical operators (chemicals)
Chemists (blank books and paper products; blast furnaces; chemicals; dyestuffs, ink, etc.; dental supplies; electrical machinery; lime, cement, and artificial stone)
Cherry processors (other foods)
Cleaners (brass factories; foundries; tin and enameled ware)
Coil solderers (electrical machinery)
Control men (foundries)
Crane men (blast furnaces)
Dental technicians (dental supplies)
Dippers (aluminum products; brass factories; other mfg. plants)
Dryers (brass factories)
Dyers (cotton cloth; dry cleaning and dyeing; textile dyeing, and finishing; woolens and worsteds)
Electricians (automobile factories; blast furnaces)
Engravers (jewelry)
Etchers (blast furnaces; electrical machinery; glass factories)
Fabricators (electrical machinery)
Finishers (electrical machinery)
Flux men (blast furnaces)
Furnace tenders (foundries)
Galvanizers (brass foundries; brass factories)
Glass men (foundries; glass factories)
Hardeners (foundries)
Head blockers (blast furnaces)
Hot galvanizers (electrical machinery)
Hot tin coaters (electrical machinery)
Insulators (electrical machinery)
Jewelers (jewelry)
Kettlemen (textile dyeing and finishing)
Layout men (foundries)
Machine operators (paper and pulp mills)
Machinists (rubber tires; storage batteries)
Maintenance tinnerns (electrical machinery)
Mechanics (foundries; garages; ice)
Metal cutters (electrical machinery)
Metal pattern makers (aluminum products; foundries; other wood-working)
Metal workers (automobile factories; foundries; furniture, showcases, cabinets, etc.; garages; metal furniture)
Mixers (explosives, ammunition and fireworks; paint and varnish factories; rubber tires)
Mounters (rubber tires)
Operators (blast furnaces; cotton cloth; dyestuffs, ink, etc.)
Pasteurizers (dairy-products)
Pattern makers (foundries; pianos and organs)
Picklers (blast furnaces; brass factories; electrical machinery; electric fixtures; foundries; tin and enameled ware)
Platers (automobile factories; blast furnaces; brass factories; electric machinery; electric fixtures; electroplating; foundries; metal furniture; other manufacturing plants; pianos and organs; printing; storage batteries)
Reelers (blast furnaces)
Repairmen (electrical machinery; foundries)
Rinsers (brass factories)
Safety equipment men (storage batteries)
Service men (electrical machinery)
Sheet metal workers (blast furnaces; electrical machinery; foundries; furniture, showcases, cabinets, etc.; metal furniture; tin and enameled ware)
Silvering men (glass factories)
Solderers (electric fixtures; automobile factories; electrical machinery; foundries; furniture showcases, cabinets, etc.; other manufacturing plants; rubber tires)

Spotters (dry cleaning and dyeing; laundries)	furnaces; electroplating; foundries; match factories; planing and milling; rubber tires; storage batteries; tin and enameled ware)
Stone setters (jewelry)	Tinsmiths (glass factories; suits, coats and overalls)
Switch assemblers (electrical machinery)	Washers (dry cleaning and dyeing; foundries)
Technicians (dairy products)	Water treaters (petroleum refineries)
Template makers (rubber tires)	Wire drawers (blast furnaces)
Testers (foundries; electrical machinery)	
Tinners (aluminum products; blast	

INDUSTRIAL HEALTH ASPECTS

SYMPTOMS OF INDUSTRIAL POISONING:

(Chlorine) The symptoms of poisoning by chlorine gas are primarily concerned with irritation of the respiratory system. The symptoms will vary with the concentration and time during which the gas is inhaled. Sustained exposures may cause bronchitis, bronchiectasis, loss of sense of smell, loss of appetite with loss of weight, headache, giddiness, insomnia and cardiac disturbances.

(Hydrochloric Acid) Hydrochloric acid poisoning, according to McNally, is more common than by sulfuric or nitric acid (not including oxides of nitrogen). The clinical symptomatology in man includes irritation of mucous membranes; conjunctivitis; pharyngeal, laryngeal and bronchial catarrh; and dental caries. The occasional occurrence of unusually dry hydrochloric acid gas under some conditions of atmospheric heat and absence of moisture, explains the increased degree of toxicity which this gas sometimes exhibits.

As previously mentioned, the possibility of the production of the very poisonous gas, arseniuretted hydrogen or arsine, should not be overlooked whenever metals are treated with hydrochloric or sulfuric acid, as in the "pickling of iron" by means of acid. Either acids or metals may contain arsenic as an impurity, and arsine is liberated when an acid acts upon a metal with the evolution of nascent hydrogen in the presence of a soluble compound of arsenic. Such conditions are common in industry and the poisonings resulting may mistakenly be attributed to acid fumes.

CYANIDES

OCCUPATIONS:

Apprentice boys (jewelry)	Buffers (metal furniture; optical goods)
Assemblers (suits, coats, and overalls)	Case hardeners (foundries; electrical machinery)
Bag hangers (fertilizer factories)	Checkers (fertilizer factories)
Bag printers (fertilizer factories)	Chemical workers (chemicals)
Barrel burnishers (other manufacturing plants)	Chemists (electrical machinery)
Blacksmiths (foundries; electrical machinery; storage batteries)	Cleaners (brass factories; copper factories; electrical machinery)
Bleachers (rayon and artificial silk)	

Cranemen (fertilizer factories)	Laborers (fertilizer factories)
Die makers (foundries)	Machine operators (foundries)
Dippers (rubber tires)	Machinists (electrical machinery)
Driers (electrical machinery)	Maintenance men (foundries; electrical machinery)
Electroplaters (other metal; automobile factories; storage batteries; electrical machinery; electroplating; electric fixtures; foundries; potteries; brass factories; clock and watch factories; glass factories; jewelry; metal furniture; suits, coats, and overalls; printing; explosives, ammunitions, and fireworks; other manufacturing plants)	Managers (foundries; electroplating)
Enamellers (electrical machinery)	Mechanics (printing)
Foremen (rayon and artificial silk; foundries; metal furniture; suits, coats, and overalls)	Meter repairmen (storage batteries)
Heat treaters (electrical machinery; car and railroad shops; automobile factories; electric fixtures; brass foundries; other metal; printing)	Metallurgists (foundries)
	Mixer-weighers (fertilizer factories)
	Picklers (tin and enameled ware; foundries)
	Punch press operators (foundries)
	Spray painters (foundries)
	Superintendents (fertilizer factories; foundries)
	Tool makers (brass factories)
	Weighers (fertilizer factories)
	Welders (foundries)

INDUSTRIAL HEALTH ASPECTS

Cyanide compounds produce in general similar symptoms. Acute cyanide asphyxia is one of the most rapid causes of death with the victim falling dead almost immediately. They are true protoplasmic poisons in that they arrest the activity of all forms of living matter.

There is a toxic effect on the central nervous system as shown by signs of paralysis and local action on peripheral nerve endings, etc. Alkaline cyanides in addition are caustic to the skin; itching, papules and vesicles which become infected are frequent. Ulcers may result.

MODES OF ENTRANCE:

All channels, even through the unbroken skin and mucous membranes. The most important path of entry of the gaseous vapors is by the respiratory tract.

SYMPTOMS OF INDUSTRIAL POISONING:

(Hydrocyanic acid). The acid and gas exert a gradual but different action; the acid is two to five times as powerful as the gas. Inversely, the gas has a stronger local irritating action on the skin and mucous membranes and sets up a distressing anesthesia. Cerebral troubles resulting from the acid are more serious than those caused by the gas. In large doses asphyxia is nearly instantaneous, with a cold sweat, dilation of pupils, eyes glassy and staring, loss of consciousness, panting, collapse and death.

In *acute poisoning*, there occurs vertigo, headache, confusion, congestion of head, oppression, palpitation, irritation, dryness and constriction of throat, dyspnea, nausea and vomiting. Later, there follows shivering,

sweating, slower pulse, convulsion, abolition of reflexes, involuntary micturition, loss of consciousness, coma with death usually resulting. In less severe cases, recovery from the feeling of constriction in the chest, weakness, unsteady gait, headache, speech difficulties and drowsiness may occur in a few days.

In *subacute poisoning*, symptoms are observed such as cough, lassitude, dyspnea, headache, backache, weakness, feeble and rapid pulse, accelerated, irregular and labored breathing, pain in chest and back, vomiting, muscular pain and cramps, trembling, paralysis, disturbances of nervous system. The breath often has the odor of bitter almonds. Color may be pale at first, then red; cyanide rash may be present.

Chronic poisoning is rare; its existence is denied by some authors. However, symptoms of chronic poisoning have been suggested by some as follows: Headache, vertigo, malaise, feeling of lassitude and weakness; unsteady gait, nausea, vomiting, loss of appetite, disorder of the gastrointestinal functions; albuminuria; suppression of tendon reflexes; disorders and irritation of the throat and respiratory system; diminution in cardiac activity, with weak pulse, palpitations and faintings; and diminution in the sensitiveness of the skin.

ELIMINATION:

Hydrocyanic acid is partly eliminated by the lungs as unchanged cyanides, traces in the sweat, and rarely by the urine.

HYDROGEN SULFIDE

OCCUPATIONS:

Occupations in which there may be contact with hydrogen sulfide are:

Alkali-salt makers	Hydrochloric-acid makers
Artificial silk makers	Maintenance men (rayon and artificial silk)
Barium carbonate workers	Match factory workers
Blast furnace workers	Mechanics (rayon and artificial silk)
Bottlers (mineral water)	Millmen (rubber)
Bronzers	Miners
Cable splicers	Mixers (rubber)
Carbon-disulfide makers	Oil-flotation-plant workers
Cellulose extractors	Oil well workers
Coke-oven workers	Petroleum refiners
Cyanogen makers	Phosphorus compound makers
Digestion-house workers (paper and pulp)	Picklers (tin and enameled ware)
Dye makers	Pulp mill workers
Fat renderers	Pyrates burners
Fertilizer makers	Pyroxylin-plastics workers
Flax-rettery workers	Sewer workers
Floormen (rayon and artificial silk)	Soap makers
Foremen (rayon and artificial silk)	Soda (leblanc) makers
Gas (illuminating) workers	Sodium-sulfide makers
Glue workers	Spinners (rayon and artificial silk)
Gypsum workers	Starch makers

Sugar refiners
Sulfide makers
Sulfur-chloride makers
Sulfuric acid makers
Sulfur miners

Tannery workers
Transparent-wrapping-materials
workers
Tunnel workers
Vulcanizers

INDUSTRIAL HEALTH ASPECTS

MODES OF ENTRANCE:

Inhalation.

SYMPTOMS OF INDUSTRIAL POISONING:

Hydrogen sulfide is one of the most poisonous gases; its toxicity and rapidity of action are of the same order as hydrogen cyanide. It is a powerful asphyxiant; in large doses respiratory failure occurs in a few seconds. It is also an irritant gas exerting local action on the mucous membranes of the eyes, mouth, throat, and air passages. Conjunctivitis may be mild or severe with secretions increased, itching, smarting, vascular changes, erosion, roughness of the lids, and perversions of sight. One of the earliest warning signs is stated to be sensitiveness to all kinds of light.

Low concentrations may produce subacute symptoms as irritation of the ocular and respiratory mucous membranes, bronchitis, confusion, excitement, headache, vertigo, ataxia, colic, nausea, vomiting, brachycardia, also pulmonary edema with bronchial pneumonia as a common sequela.

Acute poisoning may cause hyperpnea and unconsciousness in a few seconds without pain or significant warning. A choking sensation may be experienced, but unconsciousness, tonic convulsions, followed by cessation of respiration may supervene before the individual can escape. The heart may beat for 5 or 10 minutes longer, during which time artificial respiration may save the victim's life. Susceptibility to the poisoning varies for individuals but is believed increased by previous attacks.

Recovery from acute poisoning is usually complete, but cases with lasting injury occur, some with permanent psychic or nervous lesions.

LEAD AND ITS COMPOUNDS

INDUSTRIES:

Ohio industries using lead and its compounds are listed as follows:

Agricultural implements	Car and railroad shops
Aircraft	Chemicals
Asphalt and roofing materials	Copper factories
Automobile factories	Cotton cloth
Blank books and paper products	Dairy products
Blast furnaces	Dyestuffs, ink, etc.
Brass factories	Electric fixtures
Brick, tile and terra cotta	Electrical machinery

Electroplating	Other woodworking
Embroidery and laces	Paint and varnish factories
Engraving, photographic work	Paper box factories
Explosives, ammunition, fireworks	Paper and pulp mills
Fertilizer factories	Patent medicine, drugs
Foundries	Petroleum refineries
Furniture, showcases, cabinets, etc.	Potteries
Garages, etc.	Printing
Glass factories	Rubber tires
Hemp, jute and linen	Shoes
Instruments	Soap factories
Jewelry	Soft beverages
Lead and zinc	Storage batteries
Metal furniture	Suits, coats and overalls
Other chemicals	Textile dyeing and finishing
Other foods	Tin and enameled ware
Other manufacturing plants	Toys and unclassified novelties
Other metal, etc.	Wood, wicker, etc.
Other rubber factories	Woolen and worsted
Other textiles	

OCCUPATIONS:

Too numerous to list, at least 400 different occupational exposures.

INDUSTRIAL HEALTH ASPECTS

MODES OF ENTRANCE:

Lead can enter the body by inhalation, by being swallowed, and, in the case of tetraethyl lead, by being absorbed through the skin.

All lead-containing compounds are potentially dangerous. The degree of danger depends primarily upon severity of the exposure or level of dosage and the duration of such exposure, and, secondarily, upon the solubility of the compound in body fluids. This solubility is influenced, to some extent, by the particle size that enters the body.

SYMPTOMS OF INDUSTRIAL POISONING:

Symptoms may be present as metallic taste, headache, vertigo, insomnia, easily fatigued, loss of appetite, particularly for breakfast, nausea and vomiting, obstinate constipation with occasional blood in stool and abdominal colic with appearances of a surgical belly although stated to be characteristic in that pressure usually gives relief. There may be loss of weight, blue line on the gums, nervousness, peripheral neuritis, weakness of grip, muscular twitching and tremors particularly of fingers or paralysis, especially of muscles used most or muscular cramps and pains. There may be ashen pallor, moderate anemia with increased number of cells exhibiting basophilic aggregations and stippling.

Symptoms may appear after a few weeks to many months under the conditions of industrial exposure. Prolonged or severe absorption of lead can cause paralysis, hallucinations of sight, optic neuritis, albuminuric retinitis, hearing loss and encephalopathy.

Lead in blood and urine, in concentrations higher than those which occur in people who have not been exposed to abnormal quantities of lead industrially or otherwise is the best proof that excessive lead is being or has been absorbed into the body.

Normal levels: Blood—0.01-0.06 mg. lead per 100 grams, average 0.03.

Urine—0.01-0.14 mg. lead per liter, average 0.03.

Levels indicative of hazardous absorption of lead:

Blood—0.08 mg. or more per 100 grams.

Urine—0.18 mg. or more per liter (when found consistently in multiple samples).

Basophilic stippling of the red cells was once believed to be an infallible diagnostic sign of lead poisoning. However, some investigators have not found them in some cases of lead poisoning, and stippling is also found in other diseases such as malarias, secondary anemias, malignancies and other forms of intoxication (benzene, carbon monoxide and aniline). An increase in the number of such cells in a worker's blood over a previous test may be a sign of possible increased absorption. But, that is not certainly so, nor does the lack of such increase give assurance that no increase in absorption has occurred.

Porphyrins may be excreted in extremely high concentrations in the urine of persons with abnormal lead absorption and incipient or frank lead intoxication. Porphyrins are also found in individuals with some infections, vitamin deficiencies, anemias, leukemias, idiopathic porphyrinuria, and other types of intoxication. Moreover, the concentration of porphyrin in the urine does not parallel the concentration of lead in the urine or in the tissues, so that they do not indicate the severity of exposure.

Lead line must be differentiated from lines of other metallic deposits and the natural coloring found in many dark skinned people. It is not commonly seen in the mouths of people having adequate dental supervision and practicing good oral hygiene.

Electromyography is of value in detecting early neuritis before it is clinically perceptible. It is estimated that about 20 percent of the muscle substance must be lost suddenly before the patient becomes aware of weakness.

PREVENTION:

1. Maintaining in-plant exposures within safe limits by an appropriate combination of measures of engineering and medical control.
2. Promotion of good personal hygiene through instruction and supervision in the use of adequate sanitary facilities.
3. Medical supervision including properly spaced laboratory tests for the determination of lead absorption. When an abnormality has developed, physical examinations and additional laboratory tests should be done to arrive at a sound clinical diagnosis for

that individual and for others having similar exposure.

A person should be removed from lead exposure even when no symptoms of any kind exist when a blood sample contains more than 0.08 mg. lead per 100 grams, or when lead is being excreted consistently in the urine in the concentration of 0.18 mg. or more per liter. Such persons can be transferred to work involving no exposure to lead and can be returned to their original work (after the conditions which induced the hazardous absorption of lead have been corrected) when the lead concentration in the blood and urine has fallen to a safe and essentially normal level.

MANGANESE

OCCUPATIONS:

Occupations in Ohio where contact with manganese was indicated are listed as follows:

Annealers (foundries)	Fillers (paint and varnish factories; glass factories; patent medicine, drugs)
Battery makers	Filter pressmen (paint and varnish factories)
Beaders (tin and enameled ware)	Firemen (brick, tile and terra cotta)
Bin room attendants (storage batteries)	Fireworks makers
Bleaching powder makers	Foremen (paint and varnish factories)
Blenders (paint and varnish factories)	Foundry laborers (brass factories)
Bluers (automobile factories)	Furnace room laborers (glass factories)
Bottom makers (blast furnaces)	Furnace tenders (brass factories; foundries)
Calico printers	Glass Mixers
Centrifugal operators (paint and varnish factories)	Glaze preparers (potteries)
Chemical engineers (paint and varnish factories)	Glazers (potteries)
Chemists (brick, tile and terra cotta; paint and varnish factories)	Grinders (other chemicals; paint and varnish; electric fixtures; storage batteries)
Chlorine makers	Handy men (paint and varnish factories)
Closers (paint and varnish factories)	Heat treaters (foundries)
Cupola tenders (foundries)	Heaters (foundries)
Decorators (potteries)	Labelers (patent medicine, drugs)
Die and tool makers (foundries)	Laboratory assistants (paint and varnish factories)
Dippers (potteries; tin and enameled ware)	Ladellers (blast furnaces; potteries; glass factories; tin and enameled ware; patent medicine, drugs)
Dry cell paste mixers (storage batteries)	Linoleum makers
Dry pan feeders (potteries)	Loaders (brick, tile, terra cotta)
Dye makers	Machine operators (electric fixtures; foundries)
Dyers	
Enamel and paint makers (paint and varnish factories; tin and enameled ware)	
Enamelers	
Fertilizer makers	
Fettlers (potteries)	

Manganese dioxide workers	Shaders (paint and varnish factories)
Match factory workers	Shake-out men (brass factories)
Melters (brass factories; foundries; glass factories)	Shipping clerks (glass factories)
Mill hands (dyestuffs, ink, etc.)	Soap makers
Mixers (foundries; other chemicals; storage batteries; paint and varnish factories; tin and enameled ware)	Sprayers (potteries; brick, tile and terra cotta)
Molders (brass factories)	Tampers (storage and batteries)
Open hearth men (blast furnaces)	Testers (paint and varnish factories)
Packers (glass factories)	Thinners (paint and varnish)
Painters (foundries; brick, tile, terra cotta; glass factories)	Tinters (paint and varnish factories)
Paint mixers (foundries; paints and varnishes)	Transfer men (brick, tile and terra cotta)
Porcelain mixers (tin and enameled ware)	Truckers (dyestuffs, ink, etc.)
Porcelain sprayers (tin and enameled ware)	Varnishers
Sealers (storage batteries)	Varnish makers (paint and varnish)
	Washers (automobile factories)
	Weighers (foundries; paint and varnish factories; dyestuffs, ink, etc.)
	Wrappers (storage batteries)
	Zinc miners

INDUSTRIAL HEALTH ASPECTS

MODES OF ENTRANCE:

Inhalation.

SYMPTOMS OF INDUSTRIAL POISONING:

The symptoms of managanese poisoning are generally associated with the inhalation of its oxides and primarily concerned with affections of the central nervous system. The effect of manganese is said to be cumulative and the symptoms usually appear only after several months of exposure. They are evidenced by a peculiar slapping gait, weakness in legs and tremors of the whole body or extremities. Other symptoms frequently observed are mask-like face, impulsive and uncontrollable laughter, disturbances of speech, languor and sleepiness, cramps and stiffness of the muscles, propulsion and retropulsion, and exaggeration of the reflexes.

It is said that manganese, unlike lead, produces no life shortening degenerations. Seriously poisoned victims are lifelong cripples and often unfit for any gainful employment. The metal apparently makes a very definite attack upon some nonvital portion of the neuromuscular system, destroys it thoroughly if there has been sufficient exposure, and leaves the victims relatively well in every other respect.

Some predisposition to poisoning seems to be necessary and the symptoms appear generally during the first two years of exposure. The illness begins rapidly and progresses in a few months to a typical and incurable condition.

A typical case is that of a 29 year old man who worked as a laborer in the ore department of a battery company, transporting, loading, and handling manganese in powdered form. After 14 months' work, he

noticed that after dumping a wheelbarrow, he had difficulty in stopping himself when he stepped back. In a week's time this became progressively worse and the man stopped work. There was no change after six months, by which time he had difficulty in walking as his gait increased, and he would fall unless caught. More recently he has developed a tremor which involves the entire right leg, although there is no tremor in the hands, head or left leg.

Progressive bulbar paralysis and amyotrophic lateral sclerosis has been reported after chronic manganese poisoning.

MERCURY AND ITS COMPOUNDS

OCCUPATIONS:

Occupations in Ohio where contact with mercury and its compounds was indicated are listed as follows:

Acetaldehyde makers	Explosive makers (explosives, ammunitions, fireworks)
Acetic-acid makers	Felt-hat makers
Acetone (synthetic) makers	Fireworks makers
Alcohol (synthetic) makers	Foreman (electrical machinery)
Amalgam makers	Fulminate mixers
Artificial-flower makers	Fur handlers
Assemblers (instruments)	Fur preparers
Assembly line girls (electrical machinery)	Glass blowers (instruments)
Assistant technicians (dairy products)	Gliders
Bakers (electrical machinery)	Hardeners (felt hats)
Barometer makers	Instrument repairmen (rubber tires)
Battery (dry) makers	Jewelry makers (jewelry)
Bench workers (dental supplies)	Laboratory workers (dental supplies; electrical machinery)
Blockers (hats and caps)	Letter foremen (electrical machinery)
Blowers (felt hats)	Letter out men (electrical machinery)
Bronzers	Lithographers
Browners (gun barrels)	Mercury-alloy makers
Brushers (felt hats)	Mercury bronzers
Calico printers	Mercury-solder workers
Cap loaders	Mercury-still cleaners
Carroters (felt hats)	Mercury-switch makers
Cartridge makers	Mirror silverers
Chief technicians (dairy products)	Mixers (felt hats)
Chlorine makers (electrolytic)	Oven tenders (electrical machinery)
Color makers	Packers (electrical machinery)
Coners (felt hats)	Painters
Cosmetic workers	Photographic workers
Cyanogen gas makers	Platers (signs)
Dental technicians (dental supplies)	Porcelain makers
Detonator cleaners	Pump boys (electrical machinery; other manufacturing plants)
Devil operators (felt hats)	Pumpers (electrical machinery)
Disinfectant makers	Refiners (metals)
Dye makers	Scratchers (instruments)
Embalmers	
Embossers	

Sealers (instruments)
Sign makers (signs)
Sole stitchers
Steel engravers
Storage-battery makers
Tannery workers

Taxidermists
Testers (electrical machinery)
Thermometer fillers (instruments)
Welders
Wood preservers
Zinc-electrode makers

INDUSTRIAL HEALTH ASPECTS

MODES OF ENTRANCE:

Inhalation, ingestion, and absorption through the skin and subcutaneous tissues. The chief mode of entrance of mercury vapor and dust is by inhalation; of the liquid compounds and solutions of mercury, by ingestion and absorption through the skin, or possible inhalation of vapors.

SYMPTOMS OF INDUSTRIAL POISONING:

Symptoms may include metallic taste, sensation of abnormal dryness of mouth and throat, stomatitis, inflammation and softness of the gums, loosening of the teeth, salivation, pain on chewing, blue line on gums, and foetid breath, speech difficulty, twisting of words.

Psychic changes are usually present such as a nervous timidity, irritability, discouragement, depression, ease of embarrassment, blushing, crying, desire for solitude, vague fears, as of ridicule or criticism, fits of unreasonableness, impatience, inability to take orders, apathy, lack of interest, loss of memory and self-confidence, despondency, and even suicidal tendencies.

Change in hand writing. There is an intention tremor which is vibratory, intermittent, in small equal strokes and rhythmical, worse with efforts to control it, on unusual movements or when movements are observed; it disappears in sleep. The tremor attacks first the eyelids, tongue, fingers, then the voluntary muscles and is usually symmetrical—"Hatter's Shakes." The central nervous system manifestations of mercurialism are due, in the opinion of some authorities, to a diffuse encephalopathy with predominance of symptoms referable to the cerebral centers most affected. Peripheral neuritis, paralysis of hands, arms and legs have also been reported. Attacks of tremors may be so great that individuals cannot feed themselves or dress quickly.

In the more severe cases there may be found headache, vertigo, and anorexia, nausea or vomiting, diarrhea which may alternate with obstinate constipation, abdominal cramps or distension, weakness and exhaustion, and pain in the muscles, bones and joints. Usually in industrial mercurialism gastrointestinal symptoms are not prominent. Urinary changes may be slight or absent in the slow chronic form of poisoning. An increased incidence of albumin in the urine and nephritis has been reported; lymphocytosis of slight degree is also stated to occur.

Inflammation may occur at the points of excretion of mercury,

thus nephritis, colitis, gastric and duodenal ulcer have been reported as the sequelae of severe poisoning. Also inflammation may occur at the openings of salivary ducts or other points of excretion.

Individual reaction to mercury varies greatly not only as to the kind and sequence of symptoms but also as to length and intensity of exposure before poisoning occurs. While chronic mercury poisoning usually results from long exposure, studies have disclosed cases of poisoning developing within several weeks or days. A few micrograms of mercury may cause symptoms in persons sensitized to the poison and women are believed to be more susceptible than men.

METHYL ALCOHOL

OCCUPATIONS:

Occupations in Ohio where contact with methyl alcohol was indicated are listed as follows:

Aldehyde pump men	Linoleum makers
Aniline-dye makers	Managers (chemicals)
Antifreeze makers	Millinery workers
Art-glass workers	Operators (dyestuffs, ink, etc.; hemp, jute, and linen)
Artificial silk makers	Painters
Assistant supervisors (chemicals)	Paint makers
Automobile painters	Paste makers (other chemicals)
Book binders	Patent-leather makers
Bronzers	Perfume makers
Brush makers	Photo-engravers
Calico printers	Photographers
Cementers (shoes)	Polishers (wood)
Chemical workers (chemicals)	Plastics workers
Chemists (other chemicals; dye-stuffs, ink, etc.)	Printers (chemicals)
Compo conveyors (shoes)	Refinishers (wood, wicker, etc.)
Driers	Soap makers
Dye makers	Shipping clerks (other chemicals)
Etchers (foundries)	Spotters (dry cleaning and dyeing)
Explosives workers	Station attendants (garages)
Feather workers	Superintendents (other chemicals)
Felt hat makers	Tinters (printing)
Firemen	Treers (shoes)
Fitters (shoes)	Truck drivers (other chemicals)
Flap stickers (shoes)	Type cleaners
Foremen (chemicals, printing)	Upholsterers
Furniture polishers	Utility men (other chemicals)
Ink makers	Vulcanizers
Japanners	Water testers (electrical machinery)
Lacquerers	Wood workers
Lasters (shoes)	

INDUSTRIAL HEALTH ASPECTS

MODES OF ENTRANCE:

Inhalation, ingestion, or through the skin.

SYMPTOMS OF INDUSTRIAL POISONING:

Locally, methyl alcohol is an irritant to the mucous membranes of eyes, nose, and respiratory system. The skin may be dry, inflamed, or eczematous. When absorbed, causes headache, weakness, vertigo, nausea and vomiting, dilated pupils, fogginess of vision, visual hallucinations, severe colic, gastric congestion, acceleration and slowing of the heart, disturbance of pulse, cold sweats, cyanosis, sighing, loss of reflexes and sensation, decreased temperature, nystagmus, sweating, delirium, convulsions, paralysis, coma, and may result in pneumonia.

Diplopia may result from paralysis of external eye muscles; inflammation, neuritis, and atrophy of the optic nerves are common, resulting in blurred vision, then blindness, which is bilateral. Blindness may come on in a few hours or in a few days; in typical cases there is often transient improvement followed by complete and permanent blindness.

Slow chronic poisoning gives no characteristic symptoms and may result in severe damage before recognized; however, the Germans believe that vague nervous symptoms, a sense of fatigue, and irritation of the mucous membranes should lead to suspicion of poisoning. McCord has shown that poisoning in animals takes place as readily through the skin and lungs as through the stomach and intestines.

ELIMINATION:

Slowly excreted in the urine and expired air, some into the stomach, and part is slowly oxidized with the formation of formic acid which will reduce Fehling's solution and may suggest a false diagnosis of diabetes.

PHENOL AND RELATED COMPOUNDS

OCCUPATIONS:

Occupations in Ohio where contact with phenol was indicated are listed as follows:

Bakelite makers	Dyers
Bench workers (dental supplies)	Electroplaters (electrical machinery)
Benzol house operators (charcoal and coke; blast furnaces)	Engineers (other chemicals)
Brewers	Etchers
Calico printers	Explosive workers
Chemical mixers (metal furniture)	Fillers (patent medicine and drugs)
Chemists (chemicals; dyestuffs, ink, etc.; metal furniture; other chemicals; patent medicine and drugs)	Firemen (other chemicals)
Coal-tar workers	Floor oil mixers (petroleum refineries)
Color grinders (other chemicals)	Foremen (printing and publishing)
Color mixers (other chemicals)	Gas (illuminating) workers
Compounders (patent medicine and drugs)	Laboratory assistants (patent medicine and drugs)
Coppersmith (copper factories)	Laboratory workers (dental supplies)
Dental technicians (dental supplies)	Laborers (dyestuffs, ink, etc.; patent medicine and drugs)
Disinfectant workers	Lamp black maker
	Machinists (petroleum refineries)

Mixers (paint and varnish factories; petroleum refineries)	Plate molders (printing and publishing)
Mucilage makers (other chemicals)	Plate trimmers (printing and publishing)
Operators (dyestuffs, ink, etc.; rubber tires)	Power makers
Packers (patent medicine and drugs)	Press operators (other chemicals)
Paint makers	Reclaimers (rubber)
Paint mixers (paint and varnish factories)	Resin (synthetic) makers
Paint removers	Serum preparer (patent medicine and drugs)
Paste makers (other chemicals)	Shipping clerks (other chemicals)
Perfume makers	Spotters (dry cleaning and dyeing)
Pharmacists (patent medicine and drugs)	Stillmen
Picric-acid makers	Superintendents (other chemicals)
Planishers (copper factories)	Varnish cookers (paint and varnish factories)
	Waxers (dental supplies)
	Wood preservers

INDUSTRIAL HEALTH ASPECTS

SYMPTOMS OF INDUSTRIAL POISONING:

(Phenol) Phenol is noted for its powerful caustic effects upon the skin and mucous membranes. When applied to the skin it causes sensations of tingling and numbness and in the concentrated form a white eschar is formed that falls off in a few days leaving a brown stain. The anesthetic properties of phenol are well known in the practice of medicine. In minute amounts it may cause an eczema which may be brought about either by actual contact with phenol containing substances or by the presence of phenol vapors in the air. When taken internally, it causes a burning pain and a corrosion of the tissues in the mouth, throat and stomach.

In addition to its local action, phenol may produce marked changes in the central nervous system. In mild cases the chief symptoms are headache, dizziness and sometimes excitement with mild delirium. In severe cases, unconsciousness intervenes, the pulse is weak and rapid, and respiration irregular. Convulsions rarely occur and death results from failure of respiration.

Since phenol is a general protoplasmic poison which enters into combination with cell proteins, it is not surprising to observe symptoms of blood degeneration. Emaciation, nephritis, jaundice and gangrene may be encountered under certain conditions. Bandages should not be applied to skin surfaces which have come in contact with phenol as this inhibits the evaporation of the phenol. Gangrene has been known to occur under these conditions.

(Cresol) Cresol is a mixture of three isomeric cresols and is frequently sold under the name of tricresol. The symptoms are essentially the same as those listed under phenol and the toxicity is approximately of

the same order. Cresol is sold in the form of a suspension in water with soap under the name of lysol.®

(**Creosote**) Creosote is a complex mixture of phenols and their ethers obtained from wood tar. The term is also applied to a similar product obtained from the distillation of coal tar. The toxic properties are similar to those of phenols, the nature and severity depending upon the specific material under consideration. Epithelioma of the skin after prolonged exposure to creosote has been reported. It is believed that acridine may be regarded as the effective irritating principle in tar, creosote and pitch which sensitizes the skin to light.

ZINC AND ITS COMPOUNDS

INDUSTRIES:

Zinc and its compounds are used in Ohio by the following types of industries or in the production of the following products:

Agricultural implements	Lime, cement, and artificial stone
Aircraft	Match factories
Aluminum products	Metal furniture
Automobile factories	Other chemicals
Blank books and paper products	Other clay, glass, and stone
Blast furnaces	Other manufacturing plants
Brass factories	Other metals
Car and railroad shops	Other rubber factories
Charcoal and coke	Other specified mines
Chemicals	Paint and varnish factories
Clock and watch factories	Patent medicine and drugs
Coal mines	Petroleum refineries
Copper factories	Pianos and organs
Dental supplies	Planing and milling
Dyestuffs and ink	Potteries
Electric fixtures	Printing
Electrical machinery	Rubber tires
Electroplating	Ship and boat building
Engraving and photographic work	Shoes
Explosives, ammunitions and fireworks	Soap factories
Foundries	Soft beverages
Garages	Storage batteries
Glass factories	Textile, dyeing and finishing
Jewelry	Tin and enameled ware
Knit goods	Toys and unclassified novelties
Lead and zinc	Trunks and suitcases
Leather belts and goods	

OCCUPATIONS:

(Too numerous to list.)

INDUSTRIAL HEALTH ASPECTS

Industrial poisoning by zinc is chiefly concerned with the inhalation of zinc oxide fumes. Inhalation of a sufficient amount of these fumes

gives rise to a condition popularly known as "brass founders ague," "zinc chills," and "metal fume fever." After a sufficient exposure, which depends upon the individual susceptibility, there may be a slight irritation of throat. The main symptoms, however, occur later, usually several hours after the victim has left the environment where the fumes prevailed. The attack resembles "malaria chills" and may last for several hours with ordinary febrile symptoms such as lassitude, headache, nausea, muscle cramps and joint pains, and constricting sensations over the lungs.

The ingestion of zinc compounds such as zinc sulfate, zinc chloride, etc., does not have important industrial significance. Soluble zinc salts are sometimes employed as emetics and locally as astringents and antiseptics. Zinc stearate powder is an ingredient in some dusting powders. Fatal pneumonia has resulted from the inhalation of this compound.

Industrial health hazards may also prevail which are attributable to certain impurities rather than zinc itself. Arsenic, lead, and cadmium, all of which are more toxic than zinc, are frequently associated with it. Arsine poisoning is frequently caused by the treatment of zinc galvanized materials with acid.

APPENDIX II

REPORT OF COMMITTEE ON THRESHOLD LIMIT VALUES AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (1957)

RECOMMENDED VALUES

GASES AND VAPORS

	P.P.M.*	Approx. Mg. per Cu.M.†
Acetaldehyde -----	200	360
Acetic acid -----	10	25
Acetic anhydride -----	5	20
Acetone -----	1,000	2,400
Acrolein -----	0.5	1.2
Acrylonitrile -----	20	45
Allyl alcohol -----	5	12
(x) Allyl chloride -----	5	15
Allyl propyl disulfide -----	2	12
Ammonia -----	100	70
Amyl acetate -----	200	1,050
Amyl alcohol (isoamyl alcohol) -----	100	360
Aniline -----	5	19
Arsine -----	0.05	0.2
Benzene (benzol) -----	25	80

	P.P.M.*	Approx Mg. per Cu.M.†
Benzyl chloride -----	1	5
Bromine -----	1	7
Butadiene (1,3-butadiene) -----	1,000	2,200
Butanone (methyl ethyl ketone) -----	250	740
Butyl acetate (<i>n</i> -butyl acetate) -----	200	950
Butyl alcohol (<i>n</i> -butanol) -----	100	300
Butylamine -----	5	15
Butyl cellosolve (2-butoxyethanol) ----	50	240
Carbon dioxide -----	5,000	9,000
Carbon disulfide -----	20	60
Carbon monoxide -----	100	110
Carbon tetrachloride -----	25	160
Cellosolve (2-ethoxyethanol) -----	200	740
Cellosolve acetate (2-ethoxyethyl acetate)	100	540
Chlorine -----	1	3
Chlorine trifluoride -----	0.1	0.4
Chlorobenzene (monochlorobenzene) --	75	350
Chloroform (trichloromethane) -----	100	490
1-Chloro-1-nitropropane -----	20	100
(x) Chloropicrin -----	1	7
Chloroprene (2-chloro-1,3-butadiene) --	25	90
Cresol (all isomers) -----	5	22
Cyclohexane -----	400	1,400
Cyclohexanol -----	100	410
Cyclohexanone -----	100	400
Cyclohexene -----	400	1,350
Cyclopropane -----	400	690
(x) Decaborane -----	.05	0.3
Diacetone alcohol (4-hydroxy-4-methyl-2-pentanone)	50	240
Diborane -----	0.1	0.1
<i>o</i> -Dichlorobenzene -----	50	300
Dichlorodifluoromethane -----	1,000	4,950
1,1-Dichloroethane -----	100	400
1,2-Dichloroethane (ethylene dichloride) -----	100	400
1,2-Dichlorethylene -----	200	790
Dichloroethyl ether -----	15	90
Dichloromonofluoromethane -----	1,000	4,200
1,1-Dichloro-1-nitroethane -----	10	60
Dichlorotetrafluoroethane -----	1,000	7,000
Diethylamine -----	25	75
Difluorodibromomethane -----	100	860
Diisobutyl ketone -- -----	50	290

	P.P.M.*	Approx Mg. per Cu.M.†
Dimethylaniline (N-dimethylaniline)---	5	25
Dimethylsulfate -----	1	5
Dioxane (diethylene dioxide)-----	100	360
Ethyl acetate -----	400	1,400
(x) Ethyl acrylate -----	25	100
Ethyl alcohol (ethanol) -----	1,000	1,900
Ethylamine -----	25	45
Ethylbenzene -----	200	870
Ethyl bromide -----	200	890
Ethyl chloride -----	1,000	2,600
Ethyl ether -----	400	1,200
Ethyl formate -----	100	300
Ethyl silicate -----	100	850
Ethylene chlorohydrin -----	5	16
Ethylenediamine -----	10	30
Ethylene dibromide (1,2-dibromoethane)	25	190
Ethylene imine -----	5	9
Ethylene oxide -----	50	90
Fluorine -----	0.1	0.2
Fluorotrichloromethane -----	1,000	5,600
Formaldehyde -----	5	6
(x) Furfural -----	5	20
Gasoline -----	500	2,000
Heptane (<i>n</i> -heptane) -----	500	2,000
Hexane (<i>n</i> -hexane) -----	500	1,800
Hexanone (methyl butyl ketone) ----	100	410
Hexone (Methyl isobutyl ketone) --	100	410
Hydrazine -----	1	1.3
Hydrogen bromide -----	5	17
Hydrogen chloride -----	5	7
Hydrogen cyanide -----	10	11
Hydrogen fluoride -----	3	2
Hydrogen peroxide, 90% -----	1	1.4
Hydrogen selenide -----	0.05	0.2
Hydrogen sulfide -----	20	30
Iodine -----	0.1	1
Isophorone -----	25	140
Isopropylamine -----	5	12
Mesityl oxide -----	50	200
Methyl acetate -----	200	610
Methyl acetylene -----	1,000	1,650
(x) Methyl acrylate -----	10	35
Methyl alcohol (methanol) -----	200	260
Methyl bromide -----	20	80

	P.P.M.*	Approx Mg. per Cu.M.†
Methyl cellosolve (2-methoxyethanol)	25	80
Methyl cellosolve acetate (ethylene glycol monomethyl ether acetate)	25	120
Methyl chloride	100	210
Methylal (dimethoxymethane)	1,000	3,100
Methyl chloroform (1,1,1-trichloroethane)	500	2,700
Methylcyclohexane	500	2,000
Methylcyclohexanol	100	470
Methylcyclohexanone	100	460
Methyl formate	100	250
Methyl isobutyl carbinol (methyl amyl alcohol)	25	100
Methylene chloride (dichloromethane)	500	1,750
Naphtha (coal tar)	200	800
Naphtha (petroleum)	500	2,000
Nickel carbonyl	0.001	0.007
(x) Nitric acid	5	25
p-Nitroaniline	1	6
Nitrobenzene	1	5
Nitroethane	100	310
Nitrogen dioxide	5	9
Nitroglycerin	0.5	5
Nitromethane	100	250
2-Nitropropane	50	180
Nitrotoluene	5	30
Octane	500	2,350
Ozone	0.1	0.2
Pentane	1,000	2,950
Pentanone (methyl propyl ketone)	200	700
Perchloroethylene (tetrachloroethylene)	200	1,350
Phenol	5	19
Phenylhydrazine	5	22
Phosgene (carbonyl chloride)	1	4
Phosphine	0.05	0.07
Phosphorus trichloride	0.5	3
Propyl acetate	200	840
Propyl alcohol (isopropyl alcohol)	400	980
Propyl ether (isopropyl ether)	500	2,100
Propylene dichloride (1,2-dichloropropane)	75	350
Propylene imine	25	60
Pyridine	10	30
Quinone	0.1	0.4

	P.P.M.*	Approx Mg. per Cu.M.†
Stibine -----	0.1	0.5
Stoddard solvent -----	500	2,900
Styrene monomer (phenylethylene) --	100	420
Sulfur dioxide -----	5	13
Sulfur hexafluoride -----	1,000	6,000
Sulfur monochloride -----	1	6
Sulfur pentafluoride -----	0.025	0.25
p-Tertiarybutyltoluene -----	10	60
1,1,2,2-Tetrachloroethane -----	5	35
(x) Tetrahydrofuran -----	200	590
Tetranitromethane -----	1	8
Toluene (toluol) -----	200	750
o-Toluidine -----	5	22
Trichloroethylene -----	200	1,050
Trifluoromonobromomethane -----	1,000	6,100
Turpentine -----	100	560
Vinyl chloride (chloroethylene) -----	500	1,300
Xylene (Xylol) -----	200	870

* Parts of vapor or gas per million parts of air by volume.

† Approximate milligrams per cubic meter of air.

(x) These values appeared on the tentative list for 1956.

TOXIC DUSTS, FUMES, AND MISTS

Substance	Mg. per Cu. M.‡
Aldrin (1,2,3,4,10,10-hexachloro-1,4,4a,5,8,8a-hexahydro-1,4,5,8-dimethanonaphthalene) -----	0.25
Ammate (ammonium sulfamate) -----	15
Antimony -----	0.5
(x) ANTU (alpha naphthyl thiourea) -----	0.3
Arsenic -----	0.5
Barium (soluble compounds) -----	0.5
Cadmium oxide fume -----	0.1
(x) Calcium arsenate -----	0.1
Chlordane (1,2,4,5,6,7,8,8-octachloro-3a,4,7,7a-tetrahydro-4,7-methanoindane) -----	2
(x) Chlorinated camphene, 60% -----	0.5
Chlorinated diphenyl oxide -----	0.5
Chlorodiphenyl (42% chlorine) -----	1
(x) Chlorodiphenyl (54% chlorine) -----	0.5
Chromic acid and chromates (as CrO ₃) -----	0.1
Crag herbicide (sodium 2-((2,4-dichlorophenoxy)) ethanol hydrogen sulfate) -----	15

Substance	Mg. per Cu. M.‡
Cyanide (as CN) -----	5
2,4-D (2,4-dichlorophenoxyacetic acid) -----	10
(x) DDT (2,2-bis ((p-chlorophenyl))-1,1,1-trichloroethane) -----	1
Dieldrin (1,2,3,4,10,10-hexachloro-6,7,-epoxy- 1,4,4a,5,6,7,8a-octahydro-1,4,5,8- dimethanonaphthalene) -----	0.25
(x) Dinitrobenzene -----	1
Dinitrotoluene -----	1.5
Dinitro- <i>o</i> -cresol -----	0.2
EPN (O-ethyl O- <i>p</i> -nitrophenyl thionobenzenephosphonate) -----	0.5
(x) Ferbam (ferric dimethyl dithiocarbonate) -----	15
Ferrovanadium dust -----	1
Fluoride -----	2.5
(x) HETP (hexaethyl tetraphosphate) -----	0.1
Hydroquinone -----	2
Iron oxide fume -----	15
Lead -----	0.2
(x) Lead arsenate -----	0.15
Lindane (hexachlorocyclohexane, gamma isomer) -----	0.5
Magnesium oxide fume -----	15
Malathion (O-O-dimethyl dithiophosphate of diethyl mercaptosuccinate) -----	15
Manganese -----	6
Mercury -----	0.1
Mercury (organic compounds) -----	0.01
Methoxychlor (2,2-di- <i>p</i> -methoxyphenyl-1,1,1- trichloroethane) -----	15
Molybdenum (soluble compounds) -----	5
(insoluble compounds) -----	15
(x) Nicotine -----	0.5
Parathion (O,O-diethyl O- <i>p</i> -nitrophenyl thiophosphate) -----	0.1
Pentachloronaphthalene -----	0.5
Pentachlorophenol -----	0.5
Phosphorus (yellow) -----	0.1
Phosphorus pentachloride -----	1
Phosphorus pentasulfide -----	1
Picric acid -----	0.1
(x) Pyrethrum -----	2
(x) Rotenone -----	5
Selenium compounds (as Se) -----	0.1
Sodium hydroxide -----	2
(x) Sodium fluoroacetate (1080) -----	0.1
(x) Strychnine -----	0.15

Substance	Mg. per Cu. M.‡
Sulfuric acid -----	1
TEDP (tetraethyl dithionopyrophosphate) -----	0.2
TEPP (tetraethyl pyrophosphate) -----	0.05
Tellurium -----	0.1
Tetryl (2,4,6-trinitrophenylmethylnitramine) -----	1.5
(x) Thiram (tetramethyl thiuram disulfide) -----	5
(x) Thallium (soluble compounds) -----	0.1
Titanium dioxide -----	15
Trichloronaphthalene -----	5
Trinitrotoluene -----	1.5
Uranium (soluble compounds) -----	0.05
(insoluble compounds) -----	0.25
Vanadium	
(V ₂ O ₅ dust) -----	0.5
(V ₂ O ₅ fume) -----	0.1
(x) Warfarin [3-a acetonilybenzyl]-4- hydroxycoumarin) -----	0.5
Zinc oxide fumes -----	15
Zirconium compounds (as Zr) -----	5

Radioactivity: For permissible concentrations of radioisotopes in air, see "Maximum Permissible Amounts of Radioisotopes in the Human Body and Maximum Permissible Concentrations in Air and Water," Handbook 52, U.S. Department of Commerce, National Bureau of Standards, March 1953. In addition, see "Permissible Dose from External Sources of Ionizing Radiation," Handbook 59, U.S. Department of Commerce, National Bureau of Standards, Sept. 24, 1954.

Revision of the two publications mentioned above is in progress.

‡ Milligrams of dust, fume, or mist per cubic meter of air.

(x) These values appeared on the tentative list for 1956.

MINERAL DUSTS

Substance	M.P.P.C.F.**
Aluminum oxide -----	50
Asbestos -----	5
Dust (nuisance, no free silica) -----	50
Mica (below 5% free silica) -----	20
Portland cement -----	50
Talc -----	20
Silica	
high (above 50% free SiO ₂) -----	5
medium (5 to 50% free SiO ₂) -----	20
low (below 5% free SiO ₂) -----	50

Silicon carbide -----	50
Soapstone (below 5% free SiO ₂) -----	20

** Millions of particles per cubic foot of air.

TENTATIVE VALUES

Substance	P.P.M.#	Approx. Mg. per Cu. M.+
Acetylene tetrabromide -----	1	14
Allyl glycidyl ether (AGE) -----	10	45
Beryllium -----		0.002
Boron trifluoride -----	1	3
<i>n</i> -Butyl glycidyl ether (BGE) -----	50	270
(x) Butyl mercaptan -----	10	35
Chloroacetaldehyde -----	1	3
(x) Chlorobromomethane (ClBrCH ₂) -----	400	2,100
Diglycidyl ether (DGE) -----	10	55
Dimethyl formamide -----	20	60
Dipropyleneglycolmethylether -----	100	600
(x) Ethyl mercaptan -----	250	640
(x) Furfuryl alcohol -----	50	200
Glycidol -----	50	150
Isopropyl glycidyl ether (IGE) -----	50	240
Lithium hydride -----		0.025
(x) Methyl mercaptan -----	50	100
Alpha Methyl styrene -----	100	480
Monomethyl aniline -----	2	9
Paradichlorobenzene -----	75	450
(x) Perchloromethyl mercaptan -----	0.1	0.7
Phenyl glycidyl ether (PGE) -----	50	310
Propylene oxide -----	100	240
Tertiary butyl alcohol -----	100	300
(x) Toluene-2,4-diisocyanate -----	0.1	0.7
Triethyl amine -----	25	100
Vinyl toluene -----	100	480
Yttrium and inorganic compounds -----		5
Xylidine -----	5	25
(*) Teflon decomposition products -----	0	0
(*) Pentaborane (B ₅ H ₉) -----	0	0

(*) Until more data are forthcoming, it is important that atmospheric concentrations of these materials to which workers are exposed must be kept as near 0 as possible.

Parts of vapor or gas per million parts of air by volume.

+ Approximate milligrams per cubic meter of air.

(x) These values appeared on the tentative list for 1956.